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RESPIRATORY CARE
ANNIVERSARY

April 1996
Volume 41, Number 4
ISSN 0020-1324-RECACP

RESPIRATORY CARE

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Part I

- The Current Status of PEEP
- Tracheal Gas Insufflation
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A Monthly Science Journal. Established 1956. Official Journal of the American Association for Respiratory Care.

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RESPIRATORY CARE (ISSN 0020-1324) is published monthly by Daedalus Enterprises Inc, at 11030 Ables Lane, Dallas TX 75229-4593, for the American Association for Respiratory Care. One volume is published per year beginning each January. Subscription rates are $65 per year in the US; $80 in all other countries (for airmail, add $84).
The contents of the Journal are indexed in Hospital and Health Administration Index, Cumulative Index to Nursing and Allied Health Literature, and Excerpta Medica. Abridged versions of RESPIRATORY CARE are also published in Italian and Japanese, with permission from Daedalus Enterprises Inc.
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Editorials, Commentaries, and Reviews To Note

Cigarette Smoking and Health (Official Statement)—JP Hanrahan, CB Sherman, EA Bresnitz, KM Emmons, DM Mannino. Am J Respir Crit Care Med 1996;153:861-865.

Standards and Indications for Cardiopulmonary Sleep Studies in Children (Official Statement)—GM Loughlin, RT Brouillette, LJ Brooke, JL Carroll, BE Chipps, SJ England, et al. Am J Respir Crit Care Med 1996;153:866-878.

Reference Values for Pulmonary Function Tests of Japanese-American Men Aged 71 to 90 Years—DS Sharp, PL Enright, D Chiu, CM Burchfiel, BL Rodriguez, JD Curb. Am J Respir Crit Care Med 1996;153(2):805.

Pulmonary function was assessed by spirometry in 3,076 elderly Japanese-American men of the Honolulu Heart Program (HHP) cohort. The assessment was done with a stringent quality assurance program that adhered to American Thoracic Society (ATS) recommendations for spirometry. Less than 6% of the participants were unable to perform 3 acceptable spirometry maneuvers. A "healthy" subgroup of 528 men between the ages of 71 and 90 years was identified by excluding almost all smokers and subjects with lung disease and other factors negatively influencing FEV_1. Reference equations and normal ranges for FEV_1, FVC, and the FEV_1/FVC ratio were derived from the healthy group. Use of prediction equations from the Cardiovascular Health Study (CHS) of elderly European-American men consistently overpredicted FVC by 0.3 to 0.4 L and FEV_1 by 0.15 L. Men in the HHP were on average 11 cm shorter than those in the CHS. Use of a prediction equation derived from the HHP cohort when the men in the cohort were on average 22.6 years younger consistently overpredicted FEV_1 by 0.2 to 0.3 L. These results underscore the importance of using prediction equations appropriate to the ethnicity, age, and height characteristics of the subjects being studied.

Randomised Trial of Oral Morphine for Chronic Non-Cancer Pain—DE Moulin, A Iezzi, R Amireh, WKJ Sharpe, D Boyd, H Merskey. Lancet 1996;347:143.

BACKGROUND: The use of opioid analgesics for chronic noncancer pain is controversial. Some surveys report good pain relief and improvement in performance while others suggest a poor outcome with a propensity to psychological dependence or addiction. METHODS: We undertook a randomised double-blind crossover study to test the hypothesis that oral morphine relieves pain and improves the quality of life in patients with chronic regional pain of soft tissue or musculoskeletal origin who have not responded to codeine, anti-inflammatory agents, and antidepressants. Morphine was administered as a sustained-release preparation in doses up to 60 mg twice daily and compared with benztropine (active placebo) in doses up to 1 mg twice daily over 3-week titration, 6-week evaluation, and 2-week washout phases. Pain intensity, pain relief, and drug liking were rated weekly and psychological features, functional status, and cognition were assessed at baseline and at the end of each evaluation phase. FINDINGS: After dose titration in the 46 patients who completed the study, the mean daily doses of drugs were morphine 83.5 mg and benztropine 1.7 mg. On visual analogue scales, the morphine group showed a reduction in pain intensity relative to placebo in Period 1 ($p = 0.01$) and this group also fared better in a crossover analysis of the sum of pain intensity differences from baseline ($p = 0.02$). No other significant differences were detected. INTERPRETATION: In patients with treatment-resistant chronic regional pain of soft-tissue or musculoskeletal origin, 9 weeks of oral morphine in doses up to 120 mg daily may confer analgesic benefit with a low risk of addiction but is unlikely to yield psychological or functional improvement.

The Determinants of Pulmonary Diffusing Capacity in a National Sample of U.S. Adults—LM Neas, J Schwartz. Am J Respir Crit Care Med 1996;153(2):656.

Racial and gender differences in single-breath pulmonary diffusing capacity for carbon monoxide (D_{LCO}) have previously received little attention. Between 1971 and 1975, the first National Health and Nutrition Examination Survey determined D_{LCO} for 4,439 adults ages 25 to 74 residing in the United States, including 2,345 women and 438 blacks. The large sample permitted an evaluation of interactions and nonlinear relationships with D_{LCO}, and its association with biomarkers of inflammation. In a model with separate race, sex, and smoking status intercepts, positive associations with FVC, height, weight, hemoglobin concentration, and negative associations with age and neutrophil count explained 59% of the variation in D_{LCO}. In women, the decline in D_{LCO} with age was much less than in men, but after age 47 the decline with age approached that seen in men. Blacks had lower D_{LCO} levels (−1.96 mL/min/mm Hg, 95% confidence interval [CI] −0.77 to −3.14) with or without adjustment for FVC, but the changes in D_{LCO} with age and height were similar in both races. Former smokers had lower D_{LCO} (−1.13 mL/min/mm Hg, 95% CI −1.57 to −0.69), but this reduction was fully explained by the number of pack-years smoked. In current smokers, cigarettes per day and pack-years were predictive of D_{LCO} even after control for FVC and controlling for these variables fully explained the difference in D_{LCO} between never and current smokers. Peripheral neutrophil count, a biomarker of inflammation, was associated with reduced D_{LCO}. Thus, substantial sex and race differences exist for D_{LCO} within the general United States population.

The Effect of Inhaled Nitric Oxide in Pediatric Asthma—KD Pfeffer, G Ellison, D Robertson, RW Day. Am J Respir Crit Care Med 1996; 153(2):747.

Nitric oxide (NO) appears to play an important role in regulating several biologic functions in the lung, including modulation of pulmonary arterial and bronchial smooth muscle tone. Recent studies have shown that relatively high concentrations of inhaled NO reduce the bronchoconstrictor effect of methacholine in animal

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models. This raises the possibility that NO inhalation might have therapeutic potential as an alternative bronchodilator. Although investigation of this potential in adults with airway reactivity or bronchial asthma has been reported, data are lacking on the role of NO in the pediatric asthma population. We therefore performed spirometry on 12 children with asthma (mean age 11.1 years) at baseline (B), immediately after inhaling 40 ppm NO (NO-1), 10 minutes after inhaling NO (NO-10), and after inhalation of a standard β-2 agonist, albuterol (A). Baseline pulmonary functions (% predicted ± SEM) were FVC of 103.2 ± 5.6, FEV_1 of 82.2 ± 3.3, FEF_{max} of 97.0 ± 3.6, and $FEF_{25\text{-}75\%}$ of 53.5 ± 3.3. There were no statistically significant differences between baseline and NO-1 or NO-10 between any of the 4 pulmonary function parameters measured. Inhaled albuterol, however, resulted in significant improvement (% predicted ± SEM) in FVC to 109.8 ± 3.5, FEV_1 to 99.7 ± 2.9, FEF_{max} to 106.5 ± 5.1, and $FEF_{25\text{-}75\%}$ to 84.4 ± 6.4 compared with the baseline and NO inhalation groups. We conclude that NO inhaled at 40 ppm has no apparent bronchodilatory effect in pediatric subjects with asthma and mild airways disease. The clinical application of this gas as a therapeutic modality under these conditions is questionable.

Bedside Prediction of Mortality from Bacteremic Sepsis: A Dynamic Analysis of ICU Patients—D Pittet, B Thiévent, RP Wenzel, N Li, R Auckenthaler, PM Suter. Am J Respir Crit Care Med 1996;153(2):684.

The prognosis in patients with sepsis depends on severity of acute illness, underlying chronic diseases, and complications associated with infection. Adjusting for these factors is essential for evaluation of new therapies. The purpose of the present study was to determine variables readily identifiable at the bedside that predict mortality in intensive care unit (ICU) patients with sepsis and positive blood cultures. For a 5-year period, all patients of a surgical ICU presenting with positive blood cultures and sepsis were systematically analyzed for clinical variables and organ dysfunctions at the day of onset of sepsis and bacteremia and during the subsequent clinical course. The prognostic value of these variables was determined using logistic regression procedures. Of the 5,457 admissions to the ICU, 176 patients developed sepsis with positive blood cultures (3.2 per 100 admissions). The fatality rate was 35% at 28 days after the onset of sepsis; in-hospital mortality was 43%. Independent predictors of mortality at onset of sepsis were previous antibiotic therapy (odds ratio [OR], 2.40; 95% confidence interval [CI_{95}], 1.59 to 3.62; $p = 0.034$), hypothermia (OR, 1.43; CI_{95}, 1.04 to 2.44; $p = 0.030$), requirement for mechanical ventilation (OR, 2.97; CI_{95}, 1.96 to 4.51; $p = 0.009$), and onset-of-sepsis APACHE II score (OR, 1.21; CI_{95}, 1.13 to 1.29; $p < 0.001$). Vital organ dysfunctions developing after the onset of sepsis influenced outcome markedly. The best 2 independent prognostic factors were the APACHE II score at the onset of sepsis (OR, 1.13 per unit; CI_{95}, 1.08 to 1.17; $p = 0.0016$) and the number of organ dysfunctions developing thereafter (OR, 2.39; CI_{95}, 2.02 to 2.82; $p < 0.001$). In ICU patients with sepsis and positive blood cultures, outcome can be predicted by the severity of illness at onset of sepsis and the number of vital organ dysfunctions developing subsequently. These variables are easily assessed at the bedside and should be included in the evaluation of new therapeutic strategies.

Effect of Fluticasone Propionate on Sputum of Patients with Chronic Bronchitis and Emphysema—CG Llewellyn-Jones, TAJ Harris, RA Stockley. Am J Respir Crit Care Med 1996;153(2):616.

The effects of fluticasone propionate (FP) on sputum chemotactic activity, elastase inhibitory potential, albumin concentrations, and peripheral neutrophil function were studied in a group of patients with clinically stable, smoking-related chronic bronchitis and emphysema. Seventeen patients (50 to 75 years of age) were entered into a double-blind, placebo-controlled study of 1.5 mg inhaled FP/day for 8 weeks. Following treatment with FP the chemotactic activity of the sputum sol phase was lower than the corresponding values for the placebo group ($p < 0.01$). Values fell from a mean of 21.75 (± 1.58) during the run-in period to 18.37 (± 1.46; $p < 0.01$) after 4 weeks and 17.63 (± 1.86; $p < 0.05$) after 8 weeks treatment returning to 22.08 (± 1.26) cell/field after the washout period. The neutrophil elastase inhibitory capacity of the sputum sol phase increased ($p < 0.025$) with treatment from a mean of 0.177 μM elastase inhibited/L (± 0.05) pretreatment to 0.413 μM (± 0.054) after 4 weeks and 0.415 μM (± 0.054) after 8 weeks returning to 0.270 μM (± 0.07) after the washout period. Treatment with FP did not result in a change in the peripheral neutrophil functions studied or sputum albumin and myeloperoxidase concentrations. The results suggest that FP may play a protective role in these patients through a reduction in the chemotactic activity of lung secretions and potentially a reduction in the recruitment of neutrophils to the lung, and also by directly affecting the proteinase/antiproteinase balance, in favor of antiproteinases, within lung secretions.

Initial Experience with Partial Liquid Ventilation in Pediatric Patients with the Acute Respiratory Distress Syndrome—PG Gauger, T Pranikoff, RJ Schreiner, FW Moler, RB Hirschl. Crit Care Med 1996;24(1):16.

OBJECTIVE: Liquid ventilation with perfluorocarbon previously has not been reported in pediatric patients with respiratory failure beyond the neonatal period. We evaluated the technique of partial liquid ventilation in 6 pediatric patients with the acute respiratory distress syndrome of sufficient severity to require extracorporeal life support (ECLS). DESIGN: This study was a noncontrolled, phase I/II experimental study with a single group pretest/posttest design. SETTING: All studies were performed at a tertiary, pediatric referral hospital at the University of Michigan Medical School. PATIENTS: Six pediatric patients, from 8 weeks to 5 and one-half years of age, with severe respiratory failure requiring ECLS to support gas exchange. INTERVENTIONS: After 2 to 9 days on ECLS, perfluorocarbon was administered into the trachea until the dependent zone of each lung was filled. The initial dose administered was 12.9 ± 2.3 mL/kg (range 5 to 20). Gas ventilation of the perfluorocarbon-filled lungs (partial liquid ventilation) was then performed. The perfluorocarbon dose was repeated daily for a total of 3 to 7 days, with a cumulative dose of 45.2 ± 6.1 mL/kg (range 30 to 72.5). MEASUREMENTS & MAIN RESULTS: All measurements of native gas exchange were made during brief periods of discontinuation of ECLS and include P_{aO_2} and the alveolar-arterial oxygen gradient, $P_{(A\text{-}a)O_2}$. Static pulmonary compliance, corrected for weight, was also measured directly. The mean P_{aO_2} increased from 39 ± 6 to 92 ± 29 torr (5.2 ± 0.8 to 12.2 ± 3.9 kPa) over the 96 hours after the initial dose ($p = 0.021$ by repeated-measures analysis of variance). The average $P_{(A\text{-}a)O_2}$ decreased from 635 ± 10 to 499 ± 77 torr (84.7 ± 1.3 to 66.5 ± 10.3 kPa) over the same time period ($p = 0.059$), while the mean static pulmonary compliance (normalized for patient weight) increased from 0.12 ± 0.02 to 0.28 ± 0.08 mL/cm H_2O/kg ($p = 0.01$). All six patients survived. Complications potentially associated with partial liquid ventilation were limited to pneumothoraces in two of six patients. CONCLUSIONS: Perfluorocarbon may be safely administered into the lungs of pediatric patients with severe respiratory failure on ECLS and may be associated with improvement in gas exchange and pulmonary compliance. *See the related editorial:* ***Liquid Breathing: Stretching the Technological Envelope***—*JH Arnold. Crit Care Med 1996;24(1):4-5.*

Inspiratory Muscle Strength and Endurance in Steroid-Dependent Asthma—T Perez, L Becquart, B Stach, B Wallaert, A Tonnel. Am J Respir Crit Care Med 1996;153(2):610.

The adverse effect of long-term steroid treatment on respiratory muscle function remains controversial. We evaluated inspiratory muscle strength and endurance in steroid-dependent asthmatics in comparison with other asthmatics or with patients with chronic obstructive pulmonary disease exhibiting a comparable level of lung hyperinflation. Inspiratory muscle function was assessed by maximal inspiratory pressure (PI_{max}) and by an incremental inspiratory threshold loading test in 19 patients who had had steroid-dependent asthma (SDA) requiring a mean daily dose of 20.7 ± 0.8 mg prednisone for 5 ± 1.4 years. They were compared with 16 healthy control subjects, 30 patients with COPD, and 16 patients with non-steroid-dependent asthma (NSDA). PI_{max} as percentage of predicted values ($\%PI_{max}$) was not significantly different in patients with SDA (77 ± 5%) or NSDA (83 ± 6%) than in control subjects (93 ± 4%). In contrast, $\%PI_{max}$ was lower in patients with COPD (59 ± 4.4%) than in those with SDA or NSDA ($p < 0.05$) or the control subjects ($p < 0.0001$). A significant correlation was found between $\%PI_{max}$ and hyperinflation assessed by the FRC/TLC ratio ($r = 0.42$; $p < 0.0001$). Inspiratory endurance, defined as the ratio of maximal peak inspiratory pressure sustained for 2 minutes to individual PI_{max} ($P_{Lim\text{-}2}/PI_{max}$), was significantly lower in the SDA (43 ± 3%; $p < 0.0001$), NSDA (65 ± 4%; $p = 0.01$), and COPD (55 ± 3%; $p < 0.0001$) groups than in the control group (76 ± 2%). $P_{Lim\text{-}2}/PI_{max}$ was also lower in patients with SDA than in those with COPD ($p = 0.0073$) or NSDA ($p < 0.0001$). Hyperinflation plays a major role in inspiratory muscle dysfunction associated with obstructive lung disorders, but the finding of a significantly decreased endurance in patients with SDA when compared with patients with COPD, despite a lower level of hyperinflation in the former group, points to a deleterious effect of long-term corticosteroid treatment on inspiratory muscle function in asthmatics.

Detection of Excessive Bronchoconstriction in Asthma—WJ Gibbons, A Sharma, D Lougheed, PT Macklem. Am J Respir Crit Care Med 1996; 153(2):582.

Airway hyperresponsiveness is easily assessed by measuring the concentration or dose of an inhaled agonist that produces a defined response, eg, PC_{20} or PD_{20}. However, this measure does not assess excessive bronchoconstriction. We report the results of analyzing bronchial dose-response curves by measuring percent fall in vital capacity (ΔFVC%) as the response rather than the PC_{20}. In our analysis, ΔFVC% was measured at the PC_{20}, and therefore it was the *dependent* variable, whereas the concentration of agonist was the *independent* variable, in contrast to the usual bronchoprovocation tests in which the response is the independent variable and the dose is the dependent variable. We reasoned that a dose-dependent increase in gas trapping with histamine would detect excessive bronchoconstriction as a decrease in FVC; in contrast, PC_{20} measures only the ease of bronchoconstriction. In 10 patients with mild asthma the reproducibility of ΔFVC% when FEV_1 fell by 20%, ie, at the PC_{20} concentration of histamine, taken from a greater than 6-second FVC on an otherwise standard histamine challenge test was comparable to that of PC_{20}. In 10 healthy asymptomatic subjects there were only trivial falls (0.3%) in FVC to as much as 16 mg/mL histamine. In a retrospective study of 146 patients with mild asthma, the ΔFVC% was normally distributed (13.2 ± 5.5 SD%) and did not correlate with the number of β-2-agonist prescriptions or the PC_{20}, but it did correlate with the number of prescriptions written per month for oral prednisone ($r = 0.55$, $p < 0.02$). We conclude that ΔFVC% when FEV_1 falls by 20% is a safe method of detecting excessive bronchoconstriction, and it reveals that different asthmatics react in fundamentally different ways to the same agonist. This may be useful in detecting the asthmatic at risk for serious disease.

Ragweed Immunotherapy in Adult Asthma—PS Creticos, CE Reed, PS Norman, J Khoury, NF Adkinson Jr, CR Buncher, WW Busse, RK Bush, J Gadde, JT Li, HB Richerson, RR Rosenthal, WR Solomon, P Steinberg, JW Yunginger. N Engl J Med 1996;334(8):501.

BACKGROUND: Although allergen immunotherapy is effective for allergic rhinitis, its role in treating asthma is unclear. METHODS: We examined the efficacy of immunotherapy for asthma exacerbated by seasonal ragweed exposure. During an observation phase, adults with asthma who were sensitive to ragweed kept daily diaries and recorded peak expiratory flowrates between July and October. Those who reported seasonal asthma symptoms and medication use as well as decreased peak expiratory flow were randomly assigned to receive placebo or ragweed-extract immunotherapy in doses that increased weekly for an additional 2 years. RESULTS: During the observation phase, the mean (±SE) peak expiratory flowrate measured in the morning during the 3 weeks representing the height of the pollination season was 454 ± 20 L/min in the immunotherapy group and 444 ± 16 L/min in the placebo group. Of the 77 patients who began the treatment phase, 64 completed 1 year of the study treatment and 53 completed 2 years. During the 2 treatment years, the mean peak expiratory flowrate was higher in the immunotherapy group (489 ± 16 L/min, vs 453 ± 17 in the placebo group [$p = 0.06$] during the first year, and 480 ± 12 L/min, vs 461 ± 13 in the placebo group [$p = 0.03$] during the second). Medication use was higher in the immunotherapy group than in the placebo group during observation and lower during the first treatment year ($p = 0.01$) but did not differ in the 2 groups during the second year ($p = 0.7$). Asthma symptom scores were similar in the 2 groups ($p = 0.08$ in year 1 and $p = 0.3$ in year 2). The immunotherapy group had reduced hayfever symptoms, skin-test sensitivity to ragweed, and sensitivity to bronchial challenges and increased IgG antibodies to ragweed as compared with the placebo group; there was no longer a seasonal increase in IgE antibodies to ragweed allergen in the immunotherapy group after 2 years of treatment. Reduced medication costs were counterbalanced by the costs of immunotherapy. CONCLUSIONS: Although immunotherapy for adults with asthma exacerbated by seasonal ragweed exposure had positive effects on objective measures of asthma and allergy, the clinical effects were limited and many were not sustained for 2 years. *See the related editorial:* ***Is Immunotherapy for Asthma Worthwhile?***—*PJ Barnes. N Engl J Med 1996; 334(8):531-532.*

Worsening of Pulmonary Gas Exchange with Nitric Oxide Inhalation in Chronic Obstructive Pulmonary Disease—JA Barberà, N Roger, J Roca, I Rovira, TW Higenbottam, R Rodriguez-Roisin. Lancet 1996;347:436.

BACKGROUND: Inhalation of nitric oxide (NO) causes selective pulmonary vasodilation and improves arterial oxygenation in acute respiratory distress syndrome. But some patients do not respond or gas exchange worsens when inhaling NO. We hypothesised that this detrimental effect might be related to the reversion of hypoxic vasoconstriction in those patients where this mechanism contributes to ventilation-perfusion ($\dot{V}_A/\dot{Q}$) matching. METHODS: We studied 13 patients with advanced chronic obstructive pulmonary disease (COPD). We compared their responses to breathing room air, NO at 40 parts per million in air, and 100% O_2. Changes in pulmonary haemodynamics, blood gases, and $\dot{V}_A/\dot{Q}$ distributions were assessed. FINDINGS: NO inhalation decreased the mean (SE) pulmonary artery pressure from 25.9 (2.0) to 21.5 (1.7) mm Hg ($p = 0.001$) and P_{aO_2} from 56 (2) to 53 (2) mm Hg ($p = 0.014$). The decrease in P_{aO_2} resulted from worsening of $\dot{V}_A/\dot{Q}$ distributions, as shown by a greater dispersion of the blood-flow distribution (logSD Q) from 1.11 (0.1) to 1.22 (0.1) ($p = 0.018$). O_2 breathing reduced the mean pulmonary arterial pressure to 23.4 (2.1) mm Hg and caused greater $\dot{V}_A/\dot{Q}$ mismatch (logSD Q, 1.49 [0.1]). The intrapulmonary shunt on room air was small (2.7 [0.9]%) and did not change when breathing NO or O_2. INTERPRETATION: We conclude that in patients with COPD, in whom hypoxaemia is caused essentially by $\dot{V}_A/\dot{Q}$ imbalance rather than by shunt, inhaled NO can worsen gas exchange because of impaired hypoxic regulation of the matching between ventilation and perfusion.

Outbreak of Legionnaires' Disease among Cruise Ship Passengers Exposed to a Contaminated Whirlpool Spa—DB Jernigan, J Hofmann, MS Cetron, CA Genese, JP Nuorti, BS Fields, RF Benson, RJ Carter, PH Edelstein, IC Guerrero, SM Paul, HB Lipman, RF Breiman. Lancet 1996;347:494

BACKGROUND: Outbreaks of travel-related Legionnaires' disease present a public-health challenge since rapid, sensitive, and specific diagnostic tests are not widely used and because detection of clusters of disease among travelers is difficult. We report an outbreak of Legionnaires' disease among cruise ship passengers that occurred in April, 1994, but that went unrecognised until July,

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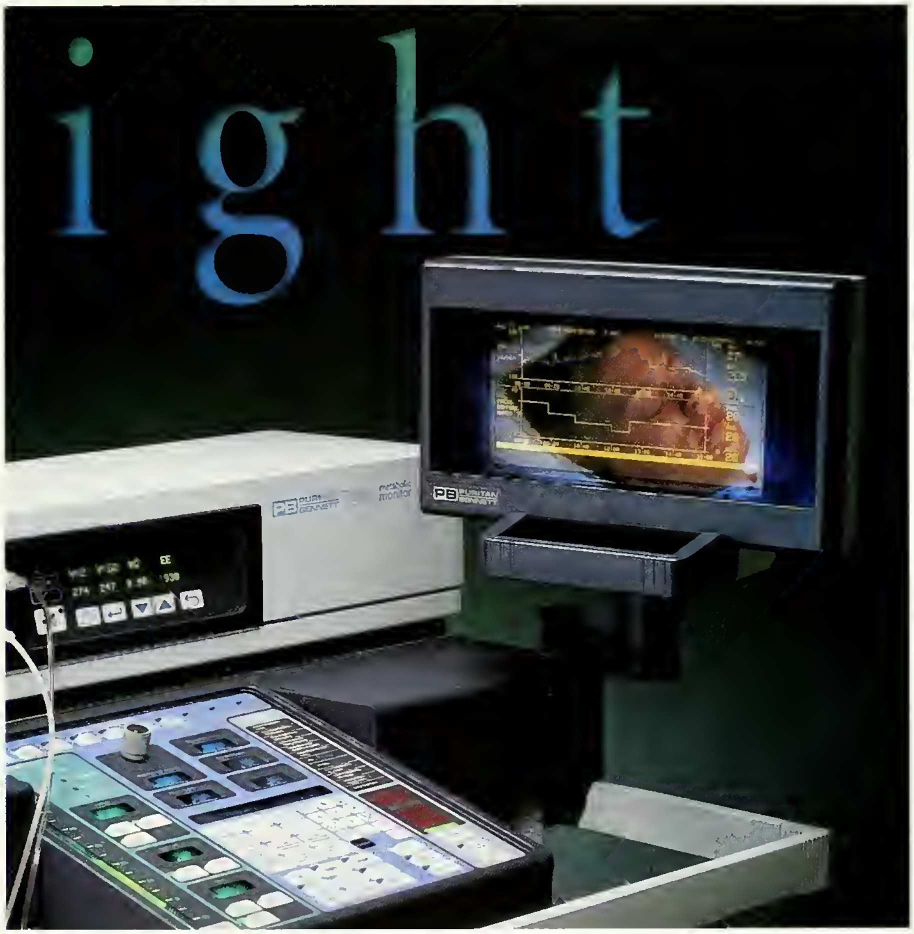

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1994. METHODS: After rapid diagnosis of Legionnaires' disease in 3 passengers by urine antigen testing, we searched for additional cases of either confirmed (laboratory evidence of infection) or probable Legionnaires' disease (pneumonia of undetermined cause). A case-control study was conducted to compare exposures and activities on the ship and in ports of call between each case-passenger and 2 or 3 matched control-passengers. Water samples from the ship, from sites on Bermuda, and from the ship's water source in New York City were cultured for legionellae and examined with PCR. FINDINGS: 50 passengers with Legionnaires' disease (16 confirmed, 34 probable) were identified from 9 cruises embarking between April 30 and July 9, 1994. Exposure to whirlpool spas was strongly associated with disease (odds ratio 16.2, 95% CI 2.8-351.7); risk of acquiring Legionnaires' disease increased by 64% (95% CI 12-140) for every hour spent in the spa water. Passengers spending time around the whirlpool spas, but not in the water, were also significantly more likely to have acquired infection. *Legionella pneumophila* serogroup 1 was isolated only from the sand filter in the ship's whirlpool spa. This isolate matched a clinical isolate from the respiratory secretions of a case-passenger as judged by monoclonal antibody subtyping and by arbitrarily primed PCR. INTERPRETATION: This investigation shows the benefit of obtaining a recent travel history, the usefulness of urine antigen testing for rapid diagnosis of legionella infection, and the need for improved surveillance for travel-related Legionnaires' disease. New strategies for whirlpool spa maintenance and decontamination may help to minimise transmission of legionellae from these aerosol-producing devices.

Cytomegalovirus: An Unexpected Cause of Ventilator-Associated Pneumonia—L Papazian, A Fraisse, L Garbe, C Zandotti, P Thomas, P Saux, G Perrin, F Gouin. Anesthesiology 1996;84 (2):280.

BACKGROUND: Cytomegalovirus (CMV) frequently is observed in immunocompromised hosts. The aim of this study was to report cases of ventilator-associated CMV pneumonia diagnosed by pathologic examination in intensive care patients without acquired immunodeficiency syndrome or hematologic malignancy or who were not receiving immunosuppressive agents. METHODS: From June 1, 1989, to May 31, 1994, 2,785 patients were hospitalized. During the study period, 60 autopsies and 26 open-lung biopsies were performed in nonimmunocompromised patients who were seen with acute respiratory failure and/or symptoms suggestive of ventilator-associated pneumonia. Cytomegalovirus pneumonia was diagnosed using pulmonary samples by the identification of large cells with large nuclei containing a basophilic or eosinophilic inclusion surrounded by a light halo. These typical findings always were associated with a diffuse interstitial pneumonitis. RESULTS: Cytomegalovirus pneumonia was diagnosed after histologic examination in 25 patients. The reason for admission to the intensive care unit was major surgery in 13 patients and medical problems in 12 patients. Ventilator-associated CMV pneumonia was diagnosed by histologic examination 22.4 ± 8.8 days after admission to the intensive care unit (median 18 days; range 10-40 days). The clinical description was similar with the 25 patients who were seen with ventilator-associated CMV pneumonia and the 61 patients without ventilator-associated CMV pneumonia. However, there was a more severe hypoxemia (72 ± 16 vs 95 ± 41 mm Hg, $p < 0.05$) and a higher Weinberg's radiologic score (9.2 ± 1.9 vs 7.4 ± 2.7, $p < 0.05$) in the ventilator-associated CMV pneumonia group. Diagnosis of ventilator-associated CMV pneumonia was made for 9 of 17 patients when shell-vial culture technique using fluorescein-labeled antibody E 13 was performed on bronchoalveolar lavage products. Four of the 8 patients treated by ganciclovir therapy died of multiple organ dysfunction syndrome. CONCLUSIONS: The diagnosis of ventilator-associated CMV pneumonia should not be excluded in intensive care patients, even those without acquired immunodeficiency syndrome, hematologic malignancy, or immunosuppressive agents on admission.

Bronchoalveolar Oxyradical Inflammatory Elements Herald Bronchopulmonary Dysplasia—M Contreras, N Hariharan, JR Lewandoski, W Ciesielski, R Koscik, JJ Zimmerman. Crit Care Med 1996;24(1):29.

OBJECTIVES: To quantify oxyradical inflammatory markers in serial endotracheal tube aspirates obtained from premature neonates at risk for developing bronchopulmonary dysplasia, and to correlate these parameters with clinical manifestations of the disease. DESIGN: Prospective cohort study. SETTING: Tertiary neonatal intensive care unit. PATIENTS: Twenty-eight intubated, premature infants, with 15 infants displaying simple respiratory distress syndrome and 13 infants eventually developing bronchopulmonary dysplasia. INTERVENTIONS: Endotracheal tube aspirates were collected and clinical severity scores were calculated longitudinally from an inception cohort during the first week of life. Diagnosis of bronchopulmonary dysplasia by standard criteria was recorded at 30 days of life. Various biochemical analyses related to pulmonary oxyradical stress were determined on endotracheal tube aspirates and were normalized according to the magnitude of serum/aspirate urea ratios. The demographic, illness severity, and biochemical characteristics of infants with simple respiratory distress syndrome and those characteristics of infants developing bronchopulmonary dysplasia were evaluated by masked comparison. MEASUREMENTS & MAIN RESULTS: Populations of respiratory distress syndrome and bronchopulmonary dysplasia infants could be differentiated during the first week of life by means of the following parameters: gestational age; birthweight; Score of Neonatal Acute Physiology; Neonatal Therapeutic Intervention Scoring System; epithelial lining fluid leukocytes; elastase; myeloperoxidase; xanthine oxidase and catalase enzyme activities; and total sulfhydryls. CONCLUSIONS: Infants with simple respiratory distress syndrome could be segregated from those infants who developed bronchopulmonary dysplasia by the magnitude of the epithelial lining fluid oxyradical inflammation markers. While infants developing bronchopulmonary dysplasia typically exhibited increased concentrations of these markers during the first week of life, those infants with simple respiratory distress syndrome displayed low, uniform, or decreasing values of these markers over this interval. Infants developing bronchopulmonary dysplasia demonstrate an early pulmonary inflammatory response, and one key aspect of this response involves various oxyradical-generating systems.

Oxygen Consumption Calculated from the Fick Equation Has Limited Utility—MC Stock, ME Ryan. Crit Care Med 1996;24(1):86.

OBJECTIVE: To determine whether oxygen consumption ($\dot{V}_{O_2}$) calculated using the Fick relationship (calculated $\dot{V}_{O_2}$) determines total body $\dot{V}_{O_2}$ accurately and precisely enough to employ this method during clinical assessment of oxygen transport. DESIGN: Methods comparison, using repeated measures during 4 physiologic states: normal heart/normal lungs, heart failure/normal lungs, normal heart/acute lung injury, heart failure/ acute lung injury. SETTINGS: University research laboratory. SUBJECTS: Thirteen adult Yucatan pigs. INTERVENTIONS: Oleic acid-induced acute lung injury; heart failure was induced with a continuous infusion of esmolol. MEASUREMENTS & MAIN RESULTS: Calculated $\dot{V}_{O_2}$ was determined by multiplying thermodilution cardiac output by the arterial-venous oxygen content difference in anesthetized, spontaneously breathing animals. Conditions were tightly controlled so that calculated $\dot{V}_{O_2}$ would be as accurate as possible. "True" $\dot{V}_{O_2}$ was measured simultaneously with a water-sealed spirometer (spirometry $\dot{V}_{O_2}$). Calculated $\dot{V}_{O_2}$ and spirometry $\dot{V}_{O_2}$ were determined and analyzed during the 4 physiologic states listed above. Pooled data also were evaluated. Mean spirometry $\dot{V}_{O_2}$, and calculated $\dot{V}_{O_2}$ differed significantly during all 4 physiologic states and when data were pooled (spirometry $\dot{V}_{O_2}$ 273 ± 70, calculated $\dot{V}_{O_2}$ 178 ± 58 mL/min, $p < 0.01$). Calculated $\dot{V}_{O_2}$ consistently underestimated spirometry $\dot{V}_{O_2}$, as demonstrated by the large, positive bias in pooled data (95 ± 59 mL/min) and in the 4 physiologic states. Linear regression of data from all 4 states yielded slopes that were indistinguishable from 1, but y-intercepts that varied from −152 to +182. For pooled data, the following equation was used: calculated $\dot{V}_{O_2} = 0.5 \times (\text{spirometry } \dot{V}_{O_2} + 46)$; $r^2 = 0.35$ Precision in pooled data was 22% of the mean spirometry $\dot{V}_{O_2}$. Data analysis for the 4 physiologic states demonstrated results similar to those results obtained when data were pooled. CONCLUSIONS: Even in a tightly controlled, clinical simulation in the laboratory, calculated $\dot{V}_{O_2}$ from the Fick relationship systematically underestimated $\dot{V}_{O_2}$ measured with a water-sealed spirometer. If true $\dot{V}_{O_2}$ changes, the magnitude and direction of change will be reflected by calculated $\dot{V}_{O_2}$ but with ≅20% error in the absolute value. Heart failure, acute lung injury, and their combination did not affect the accuracy of calculated $\dot{V}_{O_2}$. Therefore, calculating $\dot{V}_{O_2}$ using the Fick relationship is too inaccurate to be used for research purposes. Because assessment of the directional change of $\dot{V}_{O_2}$ may be clinically useful, calculated $\dot{V}_{O_2}$ can be employed with discretion during clinical oxygen transport evaluation, bearing in mind the calculation's inherent imprecision.

Evidence Based Critical Care Medicine: What Is It and What Can It Do for Us?—DJ Cook, WJ Sibbald, JL Vincent, FB Cerra, for the Evidence Based Medicine in Critical Care Group. Crit Care Med 1996;24(2):334.

OBJECTIVE: To describe the philosophy and approach to patient care called evidence based medicine, and to highlight how it can enhance the practice of intensive care. DATA SOURCES: We searched MEDLINE, reference lists, and our personal files to identify relevant literature. STUDY SELECTION: Articles on intensive care practice, critical appraisal, clinical research, and health-care delivery were selected for discussion. DATA SYNTHESIS: We summarize the rationale for evidence based medicine, its applications and future developments, and suggest several methods for intensivists to use evidence based medicine in their practice and teaching. CONCLUSIONS: Evidence based medicine can complement other foundation disciplines in intensive care. This is the first article in a series entitled "Evidence Based Critical Care Medicine" which will demonstrate how this approach can be used at the bedside.

Causes of Hypercarbia with Oxygen Therapy in Patients with Chronic Obstructive Pulmonary Disease—CW Hanson 3rd, BE Marshall, HF Frasch, C Marshall. Crit Care Med 1996;24(1):23.

OBJECTIVES: To compare data derived from a computer model of the pulmonary circulation with data from a case series of patients with chronic obstructive pulmonary disease (COPD). To evaluate the specific factors contributing to CO_2 retention due to oxygen therapy in patients with acute exacerbations of COPD. DESIGN: Data from a computer model of the pulmonary circulation were compared with a previous case series. PATIENTS: Patient data were derived from previous case series. INTERVENTIONS: Simulated application of oxygen therapy. MEASUREMENTS & MAIN RESULTS: The computer model of the pulmonary circulation generates data comparable with those data from a series of patients with COPD treated with supplemental oxygen and permits identification of the causes for hypercarbia. Therapy with supplemental oxygen alters hypoxic pulmonary vasoconstriction and modulates the Haldane effect, resulting in changes in physiologic dead space. CONCLUSION: Changes in physiologic dead space are sufficient to account for the hypercarbia developed by patients with acute exacerbations of COPD when treated with supplemental oxygen.

Evaluation of Lung Function after Intratracheal Perfluorocarbon Administration in Healthy Animals—AS Tütüncü, RM Houmes, JAH Bos, P Wollmer, B Lachmann. Crit Care Med 1996;24(2):274.

OBJECTIVES: To investigate the effects of partial liquid ventilation (ie, mechanical ventilation in combination with intratracheal administration of perfluorocarbon) on lung function, with particular attention to the integrity of the alveolocapillary membrane in healthy adult animals.

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DESIGN: Prospective, randomized, controlled study. SETTING: Laboratory at the Department of Experimental Anesthesiology, Erasmus University, Rotterdam. SUBJECTS: Ten adult male New Zealand rabbits. INTERVENTIONS: Five rabbits were intratracheally treated with 12 mL/kg of perfluorocarbon while conventional mechanical ventilation (volume-controlled, tidal volume of 12 mL/kg, respiratory rate of 30 breaths/min, inspiration-expiration ratio of 1:2, positive end-expiratory pressure of 2 cm H_2O, and an F_{IO_2} of 1.0) was applied for 3 hours. To assess the permeability of the alveolocapillary membrane, pulmonary clearance of inhaled technetium-99m-labeled diethylenetriamine penta-acetic acid (^{99m}Tc-DTPA) measurements were performed at 3 hours and compared with data from the control group ($n = 5$) treated with mechanical ventilation only, using the same ventilatory parameters. MEASUREMENTS & MAIN RESULTS: Pulmonary gas exchange and lung mechanical parameters were measured in both groups at 30-minute intervals. Mean values for P_{aO_2} in the perfluorocarbon group, although at adequate levels, were less than those values of the control group during the 3-hour study period (370 ± 44 vs 503 ± 44 torr at 3 hours [49.3 ± 5.9 vs 67.1 ± 5.9 kPa]). Peak and mean airway pressures were higher in the perfluorocarbon group (ranging from 1.9 to 3.4 cm H_2O and 0.7 to 1.3 cm H_2O, respectively) compared with the control group, while end-inspiratory airway pressure was similar in both groups. The half-life of ^{99m}Tc-DTPA was 83.7 ± 24.5 minutes in the control group, which was significantly longer ($p < 0.01$) than in the perfluorocarbon group (49.8 ± 6.1 minutes). CONCLUSIONS: These findings suggest that partial liquid ventilation with perfluorocarbons lowers pulmonary gas exchange in healthy animals, and the increased pulmonary clearance of ^{99m}Tc-DTPA after 3 hours of this type of ventilatory support may reflect minimal reversible changes in the lung surfactant system.

Total Liquid Ventilation with Perfluorocarbons Increases Pulmonary End-Expiratory Volume and Compliance in the Setting of Lung Atelectasis—R Tooley, RB Hirschl, A Parent, RH Bartlett. Crit Care Med 1996;24(2):268.

OBJECTIVE: To compare compliance and end-expiratory lung volume during re-expansion of normal and surfactant-deficient ex vivo atelectatic lungs with either gas or total liquid ventilation. DESIGN: Controlled, animal study using an ex vivo lung preparation. SETTING: A research laboratory at a university medical center. SUBJECTS: Thirty-six adult cats, weighing 2.5 to 4.0 kg. INTERVENTIONS: Heparin (300 U/kg) was administered, cats were killed, and lungs were excised *en bloc*. Normal lungs and saline-lavaged, surfactant-deficient lungs were allowed to passively collapse and remain atelectatic for 1 hour. Lungs then were placed in a plethysmograph and ventilated for 2 hours with standardized volumes of either room air or perfluorocarbon. Static pulmonary compliance and end-expiratory lung volume were measured every 30 minutes. MEASUREMENTS & MAIN RESULTS: Re-expansion of normal atelectatic lungs with total liquid ventilation was associated with an 11-fold increase in end-expiratory lung volume when compared with the increase in end-expiratory lung volume observed with gas ventilation (total liquid ventilation 50 ± 14 mL, gas ventilation 4 ± 9 mL, $p < 0.0001$). The difference was even more pronounced in the surfactant-deficient lungs with an ≅19-fold increase in end-expiratory lung volume observed in the total liquid ventilated group, compared with the gas ventilated group (total liquid ventilation 44 ± 17 mL, gas ventilation 2 ± 8 mL, $p = 0.0001$). Total liquid ventilation was associated with an increase in pulmonary compliance when compared with gas ventilation in both normal and surfactant-deficient lungs (normal: gas ventilation 6 ± 1 mL/cm H_2O, total liquid ventilation 14 ± 4 mL/cm H_2O, $p < 0.0001$; surfactant-deficient: gas ventilation 4 ± 1 mL/cm H_2O, total liquid ventilation 9 ± 3 mL/cm H_2O, $p < 0.01$). CONCLUSIONS: End-expiratory lung volume and static compliance are increased significantly following attempted re-expansion with total liquid ventilation when compared with gas ventilation in normal and surfactant-deficient, atelectatic lungs. The ability of total liquid ventilation to enhance recruitment of atelectatic lung regions may be an important means by which gas exchange is improved during total liquid ventilation when compared with gas ventilation in the setting of respiratory failure.

Cardiopulmonary Manifestations of Hantavirus Pulmonary Syndrome—GW Hallin, SQ Simpson, RE Crowell, DS James, FT Koster, GJ Mertz, H Levy. Crit Care Med 1996;24(2):252.

OBJECTIVE: To describe the clinical characteristics of a group of patients infected with the newly recognized hantavirus in the Southwestern United States. DESIGN: Case series. SETTING: Tertiary referral center. PATIENTS: All patients with confirmed hantavirus infection admitted to the University of New Mexico Hospital between May 1, 1993 and January 1, 1994. INTERVENTIONS: Records of patients with hantavirus infection were reviewed to collect all pertinent clinical data. MEASUREMENTS & MAIN RESULTS: Pulmonary disease in these patients was characterized by hypoxemia covering a wide range of severity. The cause of hypoxemia was an increased permeability (noncardiac) pulmonary edema which could be differentiated from hydrostatic (cardiac) pulmonary edema by its association with low pulmonary artery occlusion pressures and increased protein content of edema fluid. Hemodynamic measurements in severe cases showed a shock state characterized by a low cardiac index (range 1.6 to 3.0 L/min/m²), a low stroke volume index (range 10.5 to 29 mL/m²), and high systemic vascular resistance index (range 1653 to 2997 dyne · s/cm⁵ · m²). Progression to death was associated with worsening cardiac dysfunction unresponsive to treatment and causing oxygen debt and lactic acidosis. CONCLUSIONS: The 2 major life-threatening pathophysiologic changes in Hantavirus Pulmonary Syndrome are increased permeability pulmonary edema, and an atypical form of septic shock caused by myocardial depression and hypovolemia.

Inhaled Nitric Oxide in Children with Severe Lung Disease: Results of Acute and Prolonged Therapy with Two Concentrations—RW Day, M Guarín, JM Lynch, DD Vernon, JM Dean. Crit Care Med 1996;24(2):215.

OBJECTIVES: To evaluate the acute effects of 11 and 60 parts per million (ppm) inhaled nitric oxide on the pulmonary vascular resistance and systemic oxygenation of children with severe lung disease, and to compare the outcome of prolonged therapy with ≅10 and 40 ppm inhaled nitric oxide. DESIGN: Prospective, randomized study. SETTING: A 26-bed pediatric intensive care unit in a tertiary children's hospital. PATIENTS: Nineteen patients (median age 11 years, range 7 months to 16 years) with acute bilateral lung disease requiring a positive end-expiratory pressure (PEEP) of > 6 cm H_2O and an F_{IO_2} of > 0.5 for > 12 hours were treated with inhaled nitric oxide. One patient was treated twice during the same hospitalization. INTERVENTIONS: Acute hemodynamic and blood gas effects of 11 and 60 ppm inhaled nitric oxide were studied, while delivering these concentrations in random order for intervals of 20 to 30 minutes. Each interval was preceded by an interval of 20 to 30 minutes without nitric oxide. Patients were then randomized and treated for a prolonged period with ≅10 or 40 ppm inhaled nitric oxide independent of their initial acute responses to 11 and 60 ppm. Nitric oxide was discontinued when ventilatory support was decreased to a PEEP of ≤ 6 cm H_2O and an F_{IO_2} of ≤ 0.5. MEASUREMENTS & MAIN RESULTS: Inhaled nitric oxide selectively decreased pulmonary vascular resistance and improved systemic oxygenation. Acute hemodynamic and blood gas effects of 11 and 60 ppm nitric oxide were similar. Systemic oxygenation improved to a greater extent in patients with radiographic evidence of residual aerated lung regions than in patients with diffuse bilateral lung disease. Maximum methemoglobin concentrations were greater in patients treated for a prolonged period with 40 ppm nitric oxide. The mortality and duration of therapy were similar for patients treated with 10 and 40 ppm inhaled nitric oxide. CONCLUSIONS: Pulmonary vascular resistance and systemic oxygenation are acutely improved to a similar extent by 11 and 60 ppm inhaled nitric oxide, and concentrations in excess of 10 ppm are probably not needed for prolonged therapy of children with severe lung disease.

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Saturday, December 2, 1995

☐	AARC95-001a	Keynote Address. Wake Up America! *William C. Dement MD, PhD*
☐	AARC95-003	Lung Volume Reduction Surgery *Joshua O Benditt, MD*
☐	AARC95-004	Cardiology for Automated External Defibrillation Providers *William Kaye, MD*
☐	AARC95-005	Advances in the Use of High Frequency Jet Ventilation *Sherry E. Courtney, MD, MS*
☐	AARC95-006	New Modes and Expanded Capabilities of Perinatal-Pediatric Mechanical Ventilators *Mark J Heulitt, MD*
☐	AARC95-007	Advances in the Use of High Frequency Oscillatory Ventilation *Sherry E. Courtney, MD, MS*
☐	AARC95-008	Surviving a Joint Venture by Identifying the Crucial Role of the RCP on Transport Teams *Jerry A Focht, RRT*
☐	AARC95-009	Inter/Intra Hospital Transport *Jamie L. Taylor-Irizarry, RRT*
☐	AARC95-010	Flight Physiology *Mark J Heulitt, MD*
☐	AARC95-011	Air Transport of Trauma Victims *Jerry A Focht, RRT*
☐	AARC95-012	Natural History of COPD *Barry Make, MD*
☐	AARC95-013	Scientific Basis for Current Therapy *Stephen I Rennard, MD*
☐	AARC95-014	Survival Following Severe Exacerbations of COPD *Joshua O Benditt, MD*
☐	AARC95-015	Outcomes After Exacerbations of COPD (Support Study) *Alfred F Connors Jr., MD*
☐	AARC95-016	New Approaches to Treatment *Stephen I Rennard, MD*
☐	AARC95-017	Transformational Leadership *Sam P Giordano, MBA, RRT*
☐	AARC95-018	Overcoming the Barriers *John Wright, MBA, RRT*
☐	AARC95-019	Care Plan Development *John S Sabo, MS, RRT*
☐	AARC95-020	Compiling Objective Data *John Wright, MBA, RRT*
☐	AARC95-021	What Is ARDS, Who Gets It, and What Happens to Them? Has Outcome Changed? *Kenneth P Steinberg, MD*
☐	AARC95-022	What Is Appropriate Ventilator Management Today? *Neil R MAcIntyre, MD*
☐	AARC95-023	Do Corticosteroids (or Other Drugs) Alter the Course of ARDS? *Kenneth P Steinberg, MD*
☐	AARC95-024	What Happens to ARDS Survivors? *Neil R MacIntyre, MD*

Sunday, December 3, 1995

☐	AARC95-027	What Have We Learned about Lung Injury from the Management of Near-Drowning? *Jerome H Modell, MD*
☐	AARC95-028/29	RCPs Meet the Challenge of the NAEPP, Urban Message--Getting the Message Out and Breaking the Barriers to Care *Thomas J Kallstrom, RRT, Alvin V Thomas, MD*
☐	AARC95-030	School-Based Asthma Management--Strategies for Asthma Care for the Child in School & Panel Discussion *Mark W Millard, MD*
☐	AARC95-031	Management of Acute Respiratory Failure without Intubation *Robert M Kacmarek, PhD, RRT, David J Pierson, MD*
☐	AARC95-032	Changing the Paradigm: The Role of the RCP in Health Promotion/Disease Prevention *William F. Galvin, MSEd, RRT, CPFT*
☐	AARC95-033/34	RCPs in the Operating Room; RCPs on the Resuscitation Team *Richard D Branson, RRT; Charles G Durbin, Jr., MD*
☐	AARC95-035/36	RCPs in the Hyperbaric Chamber; RCPs in the Subacute Care and Skilled Nursing Facility Setting *John M Grayheal, CRTT, Dianne L Lewis, MS, RRT*
☐	AARC95-037	Laboratory and Clinical Experience in Liquid Ventilation *Ronald B Hirschl, MD*
☐	AARC95-038	Practical Considerations in the Therapeutic Administration of Inhaled Nitric Oxide *Robert M Kacmarek, PhD, RRT*
☐	AARC95-039	Tracheal Gas Insufflation *Sue A Ravenscraft, MD*
☐	AARC95-040	Ventilator Management to Avoid Barotrauma *David J. Pierson, MD*
☐	AARC95-041	Gene Therapy for Cystic Fibrosis *David E Geller, MD*
☐	AARC95-044	What Are Outcomes and Why Are They Needed? *Jeffrey A Souza, RN*
☐	AARC95-045	What Outcomes To Measure and How *Vianna Zimbel, BS, CRTT*
☐	AARC95-046a/b	Outcomes Measurement--What Lies Ahead *Jeffrey A Souza, RN* and Panel Discussion--Outcomes Measurement in Home Care
☐	AARC95-047	Guidelines for Predicting Mortality and Morbidity in Cardiac Disease *Michael B Higginbotham, MD*
☐	AARC95-048	Preoperative Assessment in Upper Abdominal and Thoracic Surgery *Charles G. Irvin, PhD*
☐	AARC95-049	Maximizing Exercise Performance in Geriatric Populations *Michael B Higginbotham, MD*
☐	AARC95-050	Mechanical Limitations During Exercise *Charles G Irvin, PhD*
☐	AARC95-051	Utilizing the AARC Clinical Practice Guidelines *Garry W Kauffman, MPA, RRT*
☐	AARC95-052	Building a Collaborative Practice Team *Dorothy M Kite-Powell, CCRN, MSN*
☐	AARC95-053	Benchmarking Your Standards of Care *Michael Boroch, MBA, RRT*
☐	AARC95-054	Building Your Outcome Management Goals *Michael Boroch, MBA, RRT*
☐	AARC95-055	Case Presentation--Outcomes Management *John S Sabo, MS, RRT*
☐	AARC95-056	Meeting the Demands of a "Reformed" Health Care System *William G Hinkle, PhD*
☐	AARC95-057	The Development of Core Curricula in Allied Health Programs *Leonard J Finocchio, MPH*
☐	AARC95-058	First Year Report on the Respiratory Care Accreditation Board (RCAB) *Thomas F Anderson, PhD, RRT*

Monday, December 4, 1995

☐	AARC95-061	Respiratory Care at the Crossroads: Evolution or Extinction? *Dean R. Hess, PhD, RRT*
☐	AARC95-062	The Impact of Restructuring on Respiratory Care Education *Crystal L Dunlevy, EdD, RRT*
☐	AARC95-063	Alternative Strategies in Clinical Education *Crystal L Dunlevy, EdD, RRT*
☐	AARC95-064	Enhanced Clinical Education in the Respiratory Care Lab *William F Galvin, MSEd, RRT, CPFT*
☐	AARC95-066	Pediatric Pulmonary Diagnostics: Aren't They Just Little Adults? *Susan Blonshine, BS, RRT, RPFT*
☐	AARC95-067	Do All Pediatric Tests Need To Comply with the ATS Standards? *Jack Wanger, MBA, RRT, RPFT*
☐	AARC95-068	Pediatric Challenges *Carl Mottram, RRT, RPFT*
☐	AARC95-069	Assessment of Respiratory Control in the Fullterm Infant *Deborah K White, BS, RRT, RPFT*
☐	AARC95-070	Scientific Rationale for Clinical Use of Nitric Oxide *John E. Thompson, RRT*
☐	AARC95-071	Current and Future Applications of Nitric Oxide *Robert M Kacmarek, PhD, RRT*
☐	AARC95-072	Watch It!--Practical Aspects of Nitric Oxide *John E Thompson, RRT*
☐	AARC95-073/74	The View of the Neonatologist, The View of the Pediatrician *Sherry E Courtney, MS, MD, David E. Geller, MD*
☐	AARC95-075/76	The View of the Anesthesiologist, The View of the Pulmonologist *Charles, G Durbin Jr., MD, Richard Sheldon, MD*
☐	AARC95-077/78	The View of the Surgeon, The View of the Intensivist *Jay A Johannigman, MD, James M Hurst, MD*
☐	AARC95-081	Respiratory Muscle Retraining--Does It Improve Function? *Barry Make, MD*
☐	AARC95-082	Metabolic Testing--Does It Change the Exercise Prescription? *Mark W Millard, MD*
☐	AARC95-083	Five Important Articles You Should Have Read on Pulmonary Rehabilitation in 1995 *Barry Make, MD*
☐	AARC95-084	Pre-Hospital Management and Transport of the Trauma Patient *Jay A Johannigman, MD*
☐	AARC95-085	Management of the Pediatric Trauma Patient *Joseph J Tepas III, MD*

☐	AARC95-086	Management of the Pregnant Trauma Patient *David T Goldsberry, MD*
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☐	AARC95-090	Meeting the Health Care Challenges of the Aging Population *Carl P Wiezalis, MS, RRT*
☐	AARC95-091	Geriatric Nutritional Assessment *Pam Z Cacchione, RN, MSN, C-GNP*
☐	AARC95-092	Enhancing Communication and Teaching Techniques for the Older Patient *Pam Z Cacchione, RN, MSN, C-GNP*
☐	AARC95-093	Managing End-Of-Life Issues *Patrick J Cacchione*
☐	AARC95-094	Physiology of Alpha 1-Antitrypsin and Pathophysiology of Deficiency States *Michael J Krowka, MD*
☐	AARC95-095	Clinical Features of Alpha 1-Antitrypsin Deficiency *James K Stoller MD*
☐	AARC95-096	Current and Future Treatment Options for Alpha 1-Antitrypsin Deficiency Augmentation Therapy, Gene Therapy *Charlie Strange, MD*
☐	AARC95-097	Lung Transplantation for Alpha 1-Antitrypsin Deficiency Emphysema *Marshall I Hertz, MD*
☐	AARC95-098	The 1995 JCAHO Standards -- The First Year's Experience *Kathleen Brinton, RRT*
☐	AARC95-099	Developing and Using an Effective Plan of Care *Vianna Zimbel, BS, CRTT*
☐	AARC95-100a	Improving Organization Performance & Patient Satisfaction *Vianna Zimbel, BS, CRTT*
☐	AARC95-100b	Questions and Answers -- Home Care The Impact of Accreditation

Tuesday, December 5, 1995

☐	AARC95-103	Equipment Validation in the Pulmonary Lab *Jack Wanger, MBA, RRT, RPFT*
☐	AARC95-105	What Features Do We Really Need in Mechanical Ventilators? *Robert M Kacmarek, PhD, RRT*
☐	AARC95-106	The Consensus Statement from the 1995 Conference on Assessing Innovations on Mechanical Ventilatory Support *Thomas D East, PhD*
☐	AARC95-107	Defining Subacute Care Services. Patients, Settings, and Outcomes *Kathleen M Griffin, PhD*
☐	AARC95-108	Using Outcome Data to Justify Subacute Care Services *Dianne L Lewis, MS, RRT*
☐	AARC95-109	Providing Subacute Services in a Managed Care Environment *Kevin A Cornish, RRT*
☐	AARC95-110	Five Things I Learned this Year about Aerosol Therapy *Jon O Nilsestuen PhD, RRT*
☐	AARC95-111	Five Things I Learned this Year about CPR *Thomas A Barnes, EdD, RRT*
☐	AARC95-112	Five Things I Learned this Year about Managed Care *Michael Boroch, MBA RRT*
☐	AARC95-113	Five Things I Learned this Year about Sleep Apnea *Karen J Stewart, BS, RRT*
☐	AARC95-114	Five Things I Learned this Year about What the RC Future Might Be (According to the Federal Government) *Cheryl A Brown, MHA*
☐	AARC95-115	Introduction and Overview. Identifying a Clinical Question and Formulating a Hypothesis *James K Stoller, MD, Shelley C Mishoe, PhD, RRT*
☐	AARC95-116/117	A Vignette What is a Null Hypothesis?; Reviewing the Literature and Finding a Mentor *Dean R Hess, PhD, RRT; Joseph L Rau, PhD, RRT*
☐	AARC95-118-120	A Vignette The p Value -- Just What Is It?; Validating Patient-Driven Protocols -- Choosing a Design, A Vignette Getting Your Abstract Accepted *Dean R Hess, PhD, RRT; Charles G Durbin Jr, MD, Kaye Weber, MS, RRT*
☐	AARC95-121	Cystic Fibrosis: It's Not Just for Kids Anymore *Stanley B. Fiel, MD*
☐	AARC95-122	Language of Managed Care *John R Walton, FACHE, RRT*
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☐	AARC95-124	Preparing for Business Ventures *John R Walton, FACHE, RRT*
☐	AARC95-125	Outcomes Management Paradigm Shift *Dorothy M Kite-Powell, RN, MSN, CCRN*
☐	AARC95-126	RCPs Should Insert Chest Tubes *Richard Sheldon, MD & Paul A Selecky, MD*
☐	AARC95-127	Waveforms and Graphics Should Be Monitored and Available on All Mechanically Ventilated Patients in the ICU *Robert S Campbell, RRT & Richard D Branson, RRT*
☐	AARC95-128	Hospitals Need RC Technical Directors *John S Sabo, MS, RRT & Michael Boroch MBA, RRT*
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☐	AARC95-130	Are RCPs Really Going To Be Able to Depend on Computers? *Thomas D East, PhD*
☐	AARC95-131	The Effects of State Licensure and Credentialing on Respiratory Home Care *Jill A Eicher, MPA*
☐	AARC95-132	Washington Update on Home Care *Cheryl A Brown, MHA*
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☐	AARC95-142	Airway Mechanics. Laboratory Measurement *Robert A Brown, BS, RRT, RPFT*
☐	AARC95-144	Bronchial Provocation Testing Relationship to Airway Mechanics *Jack Wanger MBA RRT, RPFT*
☐	AARC95-145	Assessment of Airway Mechanics Outside the Pulmonary Function Laboratory *Charles G Irvin, PhD*

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The 1996 Summer Forum, scheduled for July 12–14 in Naples, FL, will offer two opportunities for participants to share their scholarly activities with colleagues:

1. Research abstract presentations dealing with respiratory care education. (Paper presentations will be limited to 15 minutes, including five minutes for discussion.)
2. Poster presentations dealing with education models, methods, or materials that can be shared for noncommercial use. (Individual topics and presenters will be briefly introduced; additional time will be allowed for individual review of posters or display materials and interaction with the presenters.)

Research abstracts and poster presentation proposals must be submitted by April 15, 1996, for review by the Education Section Review Committee. All abstracts and proposals will be peer-reviewed, and authors will be notified of decisions by May 15. Questions may be directed to the review committee chair, Pat Munzer.

Proposals must include three components:

1. Cover sheet that includes the following information:
    a. Type of presentation (research abstract or poster presentation)
    b. Title of presentation
    c. Names, primary titles, work addresses, and day telephone numbers of the authors; the name of the author who will present the paper and receive correspondence should be listed first
    d. A statement of previous presentations or publications of the same or similar work
    e. Two true/false questions with answers
    f. Other information required by funding sources
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ABSTRACTS & POSTER PRESENTATIONS

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Type abstract double-spaced on plain white paper with one-inch margins. The abstract should be written as a single paragraph and limited to 300 words. The research abstract should include the following:

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c. Brief statement of methodology and findings
d. Conclusions
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A maximum of two pages of camera-ready charts and tables may be attached to aid in the selection of the abstract.

Poster Presentations

Type abstract double-spaced on plain white paper with one-inch margins. The abstract should be written as a single paragraph and limited to 150 words. The poster presentation abstract should include the following:

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Each manuscript should be double-checked for completeness and accuracy before it is submitted.

Spell out terms used for the first time, followed by abbreviations in parentheses, e.g., norm-referenced testing (NRT). Thereafter, the term may be abbreviated throughout.

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with David J. Pierson, MD, and Richard D. Branson, RRT
- Live Broadcast — February 20, 12:30 p.m. to 2:00 p.m.
- Teleconference — March 11, 12:30 p.m. to 1:00 p.m.

#2• *Managing the Ventilator: What and When*
with Dean R. Hess, PhD, RRT, and Richard D. Branson, RRT
- Live Broadcast — April 2, 12:30 p.m. to 2:00 p.m.
- Teleconference — April 22, 12:30 p.m. to 1:00 p.m.

#5• *Theory and Application of Neonatal Ventilation: What, When, and Why*
with Rob Chatburn, RRT, and Richard D. Branson, RRT
- Live Broadcast — August 27, 12:30 p.m. to 2:00 p.m.
- Teleconference — September 16, 12:30 p.m. to 1:00 p.m.

#7• *Airway Management: Tricks of the Trade*
with Charles G. Durbin, MD, and David J. Pierson, MD
- Live Broadcast — December 10, 12:30 p.m. to 2:00 p.m.
- Teleconference — December 19, 12:30 p.m. to 1:00 p.m.

Respiratory Care Issues

#3• *Managing Demand for Respiratory Care Services*
with James K. Stoller, MD, and Sam Giordano, MBA, RRT
- Live Broadcast — May 21, 12:30 p.m. to 2:00 p.m.
- Teleconference — June 10, 12:30 p.m. to 1:00 p.m.

#4• *Capturing Respiratory Services Outside the Hospital Part I: Home Care*
with Allan B. Saposnick, MS, RRT, and Sam Giordano, MBA, RRT
- Live Broadcast — July 9, 12:30 p.m. to 2:00 p.m.
- Teleconference — July 29, 12:30 p.m. to 1:00 p.m.

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with Kevin Cornish, RRT, and Sam Giordano, MBA, RRT
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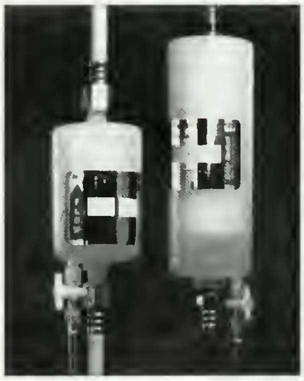

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Dear Reader:

Since 1982 the American Association for Respiratory Care and its science journal RESPIRATORY CARE have published the proceedings from state-of-the-art and/or consensus conferences organized by the journal Editorial Board. This issue contains the proceedings from the 1995 conference on *Mechanical Ventilation: Ventilatory Techniques, Pharmacology, and Patient Management Strategies*. Previous conferences covered the following:

1995 - *Resuscitation in Acute Care Hospitals* (April and May, Volume 40, Numbers 4 and 5)
1995 - *Consensus Conference on Assessing Innovation on Mechanical Ventilatory Support* (September, Volume 40, Number 9)
1994 - *Controversies in Home Respiratory Care* (April and May, Volume 39, Numbers 4 and 5)
1993 - *Oxygenation in the Critically Ill Patient* (June and July, Volume 38, Numbers 6 and 7)
1992 - *Emergency Respiratory Care* (June and July, Volume 37, Numbers 6 and 7)
1992 - *Consensus Conference on the Essentials of Mechanical Ventilators* (September, Volume 37, Number 9)
1991 - *Respiratory Care of Infants and Children* (June and July, Volume 36, Numbers 6 and 7)
1991 - *Consensus Conference on Aerosol Delivery* (September, Volume 36, Number 9)
1990 - *Noninvasive Monitoring in Respiratory Care* (June and July, Volume 35, Numbers 6 and 7)
1989 - *Pulmonary Function Testing* (June and July, Volume 34, Numbers 6 and 7)
1988 - *PEEP* (June and July, Volume 33, Numbers 6 and 7)
1987 - *Mechanical Ventilation* (June and July, Volume 32, Numbers 6 and 7)
1986 - *Neonatal Respiratory Care* (June and July, Volume 31, Numbers 6 and 7)
1985 - *Monitoring of Critically Ill Patients* (June and July, Volume 30, Numbers 6 and 7)
1984 - *Perioperative Respiratory Care* (May and June, Volume 29, Numbers 5 and 6)
1983 - *The Management of Acute Respiratory Failure* (May, Volume 28, Number 5)
1982 - *Complications of Respiratory Therapy* (April, Volume 27, Number 4)

We believe that publication of the conference proceedings is among the greatest accomplishments by the Association and the Journal. For this we owe a lot to many unselfish individuals: the Editorial Board, the speakers, the Conference chairmen, Editor Pat Brougher, and Associate Editor Kaye Weber. Their planning, presentations, writing, and editing have resulted in documents treasured by respiratory care practitioners everywhere. We are also grateful to Glaxo Wellcome for their support since 1988. Glaxo Wellcome's generous grants have allowed the Editorial Board to plan and present conferences covering topics of tremendous importance to pulmonary medicine.

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Ray Masferrer RRT
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MECHANICAL VENTILATION: VENTILATORY TECHNIQUES, PHARMACOLOGY, & PATIENT MANAGEMENT STRATEGIES

Part I

A Special Issue

based on a conference
held October 6–8, 1995
in Cancún, Mexico

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The Current Status of PEEP

William T Peruzzi MD

Introduction
Historical Perspective
Pulmonary Effects of PEEP
  Increased Alveolar Volumes
  Redistribution of Lung Water
  Alveolar Recruitment
Cardiovascular Effects of PEEP
  Decreased Venous Return
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Other Effects of PEEP
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PEEP Management Strategies
In Conclusion

Introduction

The application of positive airway pressure at end exhalation, in conjunction with positive pressure ventilation, is known as positive end-expiratory pressure (PEEP) therapy.[1] When positive pressure is applied in conjunction with spontaneous ventilation, the term continuous positive airway pressure (CPAP) is used.[2,3] Positive end-expiratory pressure is most often applied to patients with acute restrictive lung pathology to improve oxygenation and lung function. This applies to adults, children, and infants (acute lung injury, ALI; or the acute respiratory distress syndrome, ARDS) and to neonates (respiratory distress syndrome, RDS).

PEEP can be applied with devices that impede expiratory flow, such as flow resistors that allow adjustment of expiratory pressure by modification of orifice size, and threshold resistors, which may be gravity dependent (water column) or gravity independent (eg, spring-loaded or mushroom-type ventilator exhalation valves with balloons or diaphragms). The technical aspects of PEEP devices are beyond the scope of this article, but this topic has been reviewed and summarized elsewhere,[4-9] including the two issues of this Journal that emanated from the RESPIRATORY CARE Journal Conference "Positive End-Expiratory Pressure."

In this paper, I review the current status of PEEP with regard to the effects on alveolar recruitment and lung volumes, associated hemodynamic effects, effects on the outcome of acute pulmonary disease, secondary humoral and endocrinologic effects, and associated complications.

Historical Perspective

The use of PEEP, as it is currently recognized, began with the description of the adult respiratory distress syndrome by Ashbaugh, Petty, and co-workers between 1967 and 1969.[10,11] The application of PEEP was expanded by Gregory et al[12] in 1970 to include spontaneously breathing neonates with hyaline membrane disease. The goal-directed titration of PEEP

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A version of this paper was presented by Dr Peruzzi during the RESPIRATORY CARE Journal Conference, Mechanical Ventilation: Ventilatory Techniques, Pharmacology, and Patient Management Strategies, held October 6-8, 1995, in Cancún, Mexico.

Reprints: William T Peruzzi MD, Medical Director, Respiratory Care Services, Northwestern Memorial Hospital, 250 E Superior Street, Wesley Pavilion, Suite 678, Chicago IL 60611.

in patients with severe ARDS was described by Kirby, Downs, Civetta, and co-workers in 1975.[13] These authors described key treatment principles with end points that included arterial oxygenation (P_{aO_2}), inspired oxygen concentration (F_{IO_2}), physiologic shunt fraction ($\dot{Q}_{sp}/\dot{Q}_{tot}$), and hemodynamic stability. This approach, which focused on the reduction of $\dot{Q}_{sp}/\dot{Q}_{tot}$ as an end point with the intent to avoid oxygen toxicity, was termed "optimal" PEEP.[14] Other approaches to the titration of PEEP with different physiologic end points were also advanced. These included the "best" PEEP approach, which emphasized static lung compliance as the primary end point with attention to maximizing oxygen transport and minimizing dead-space ventilation.[15] Walkinshaw and Shoemaker[16] advanced the concept of "preferred" PEEP, which is that PEEP level associated with maximal oxygen consumption ($\dot{V}_{O_2}$). Titration of PEEP by monitoring the $P_{aCO_2} - P_{ETCO_2}$ difference as an indicator of dead-space ventilation was advocated as a way by which to determine "appropriate" PEEP.[17] Perhaps the most current and popular approach to PEEP therapy is that of "least" PEEP, which advocates the use of the lowest level of PEEP necessary to maintain a P_{aO_2} of 60 torr with an $F_{IO_2} \leq 0.5$ and an adequate cardiac output (C.O.).[18,19] This approach has been associated with less barotrauma and hemodynamic embarrassment when compared with other strategies that have focused on $\dot{Q}_{sp}/\dot{Q}_{tot}$ reduction to < 15% with increasing PEEP levels. The current emphasis for the ventilatory management of ARDS is focused on the avoidance of volutrauma, or ventilator-induced lung injury secondary to alveolar overdistention.[20] This has provided new ideas and generated new controversies concerning the optimal modes of ventilatory support to be used in this clinical setting, but the titration of PEEP remains an integral part of ventilator management strategy.

Pulmonary Effects of PEEP

The application of PEEP to the lung affected by acute parenchymal restrictive processes results in several important changes in the pulmonary system. It has been well documented that application of PEEP in such circumstances improves oxygenation, increases functional residual capacity (FRC), decreases $\dot{Q}_{sp}/\dot{Q}_{tot}$, and improves lung compliance. The mechanisms by which these effects occur are several.

Increased Alveolar Volumes

That alveolar volumes increase progressively as PEEP is increased from 0 to 15 cm H_2O has been demonstrated with light-incident photomicroscopy in normal rat lungs in the study by Daly et al.[21] In that study, (1) alveolar diameters increased linearly as PEEP was increased from 0 to 10 cm H_2O; (2) end-expiratory alveolar diameters increased to a greater degree than end-inspiratory diameters; (3) increases in alveolar diameter progressively diminished with levels of PEEP above 10 cm H_2O PEEP and plateaued at approximately 15 cm H_2O PEEP; (4) beyond 15 cm H_2O PEEP, alveolar pressure increased without a measurable increase in alveolar diameters. Thus, it is apparent that there are upper limits to the distensibility of normal alveoli and that, within these limits, the application of PEEP can increase FRC simply by increasing alveolar size (Fig. 1).

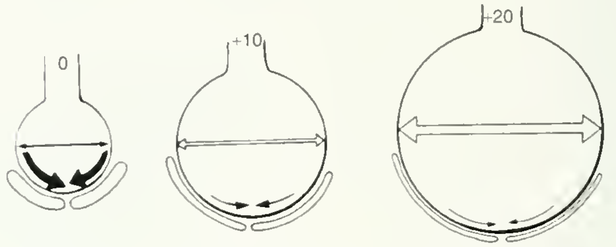

Fig. 1. Increasing alveolar volumes with 10 and 20 cm H_2O PEEP. Black arrows represent elastic forces; white arrows represent distending forces. (Reproduced, with permission, from Reference 1.)

Redistribution of Lung Water

The application of PEEP to patients with either cardiogenic or noncardiogenic pulmonary edema improves oxygenation and pulmonary mechanics largely because of the effects on the distribution of lung water. It has been established, however, that PEEP does not decrease total lung water;[22,23] rather, it appears that PEEP directly increases the accumulation of extravascular lung water.[24-26] The application of PEEP to the edematous lung decreases intra-alveolar fluid volume, increases interstitial lung water, and facilitates the movement of water from the less compliant interstitial spaces where gas exchange occurs (between the alveolar epithelium and pulmonary capillary endothelium) to the more compliant interstitial spaces (peribronchial and hilar regions).[27-29] The mechanism by which the application of PEEP brings about these changes involves immediate increases in alveolar and pulmonary vascular pressures without immediate important effects on interstitial pressures. The increased pressure gradient that occurs increases transduction of water into the interstitium of the lung. This redistribution of interstitial water, which is illustrated in Figure 2, improves oxygenation, lung compliance, and ventilation-perfusion matching when applied in either cardiogenic or noncardiogenic pulmonary edema.

Alveolar Recruitment

Alveolar recruitment is a term commonly used to refer to an increase in FRC secondary to inflation of previously collapsed alveoli.[30,31] This phenomenon occurs when PEEP is applied to lungs with appropriate pathology and plays an important role in restoring the FRC and bringing about the

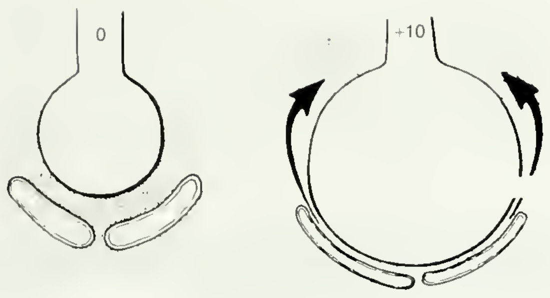

Fig. 2. Redistribution of extravascular lung water with PEEP. (Reproduced, with permission, from Reference 1.)

other beneficial effects of PEEP.[32] Studies of the effects of PEEP on volume-pressure (V-P) curves during ARDS have demonstrated an inflection point that is thought to represent the critical pressure necessary to reopen, or recruit, closed peripheral airways and/or collapsed alveolar units.[33-35] The phenomenon of alveolar recruitment with PEEP in ARDS has been studied recently in a more visually direct and quantifiable fashion by Gattinoni and co-workers.[36] These investigators have used computed tomography (CT) scanning techniques to correlate the physiologic changes observed in volume-pressure relationships with morphologic changes noted on CT scans in ARDS patients during PEEP therapy.[37] Using this technique, they have been able to calculate regional gas-tissue ratios (g/t) during titration of PEEP (Fig. 3)[38] and to determine which regions of the lung inflate without recruitment, which inflate with recruitment, and which do not inflate at all. Plots of the g/t at various PEEP levels demonstrate three different characteristic curves that correspond to different types of lung pathology and responses to PEEP (Fig. 4). Figure 4-A demonstrates a linear increase in g/t in response to PEEP titration that is consistent with progressive increases in alveolar size. Figure 4-B demonstrates a classic inflection point (P_{flex}) in the g/t curve, at a critical PEEP level, which is consistent with alveolar recruitment. Figure 4-C demonstrates no increase in g/t with increasing PEEP, which indicates no additional lung inflation and is consistent with a consolidative process.

The response to PEEP titration varies not only with the type of lung pathology but also with the region of lung studied. Figure 5 demonstrates that more dependent lung regions undergo greater degrees of alveolar recruitment, less dependent lung regions tend to experience greater increases in alveolar volume without recruitment, and regions of lung that are not responsive to PEEP (consolidated) are distributed throughout the lung. It is interesting to note that in the study of Gattinoni et al[39] when the type of PEEP responsiveness was compared with mortality, it became evident that those patients demonstrating a more linear g/t PEEP-response (alveolar expansion) had a better survival rate than those demonstrating a biphasic response (alveolar recruitment) (71 ± 4.5% vs 47 ± 5.1%, $p < 0.01$). This result might be anticipated because the degree of lung pathology is certainly more severe, and the underlying general pathophysiology is very likely worse in the latter group.

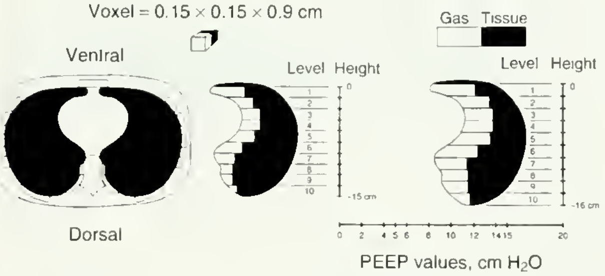

Fig. 3. Diagrammatic representation of computed tomographic scans of lungs with ARDS. The distance between the dorsal and ventral surface is divided into 10 equal lung levels. Each lung level comprises 300-500 volume elements that are the basic units of computed tomographic reconstruction (voxels). Scanning and division into lung levels is repeated at each positive end-expiratory pressure (PEEP) level. The gas-tissue ratio (g/t) of each level represents the average g/t of all the voxels composing that level. (Reproduced, with permission, from Reference 38.)

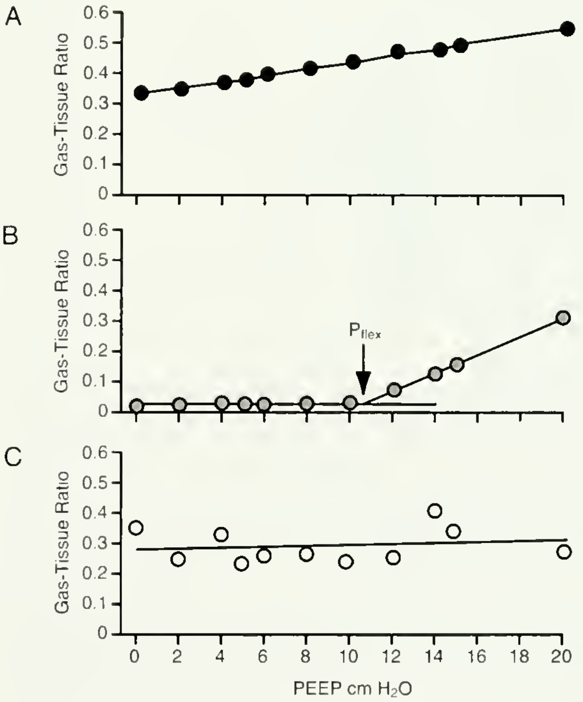

Fig. 4. Curves of gas-tissue ratios (g/t) at various PEEP levels as determined by computed tomographic scanning. A: linear g/t curve indicative of alveolar expansion; B: biphasic g/t curve with classic inflection point (P_{flex}) indicative of alveolar recruitment; C: insignificant or unchanging g/t curve indicative of alveolar consolidation. (Reproduced, with permission, from Reference 38.)

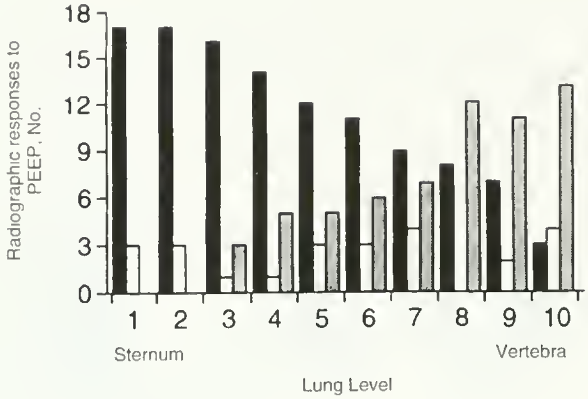

Fig. 5. The distribution of the various responses to positive end-expiratory pressure (PEEP) according to lung level as determined by computed tomography scanning. g/t-P = gas-tissue ratio PEEP curve. ■ = linear g/t-P curve; ▨ = biphasic g/t-P curve; ☐ = insignificant g/t-P curve. (Reproduced, with permission, from Reference 38.)

Alveolar collapse and recruitment also occur during normal tidal ventilation. Gattinoni et al[39] also used CT scanning methods to delineate the effects of PEEP on the distribution of ventilation and alveolar recruitment during tidal ventilation. These authors confirmed radiographically that the application of PEEP to ARDS-affected lungs results in a redistribution of tidal ventilation from the less dependent (upper) regions of lung to the more dependent (lower) regions of the lung. They also demonstrated that the application of PEEP in such clinical circumstances reduces the cyclic alveolar collapse and recruitment that occurs with normal tidal ventilation, primarily in the middle lung fields. It is interesting to note that the plateau pressures that were required to reopen or recruit collapsed middle lung regions during tidal ventilation were found to be lower than would be expected if true alveolar collapse were occurring. In light of the fact that the radiographic criteria for collapsed lung permit inclusion of lung regions that could be up to 10% occupied by gas, these middle regions of lung could possibly be undergoing recruitment partly due to re-expansion of collapsed alveoli and partly due to reopening of collapsed small airways. These data provide some evidence to support the purported effects of PEEP on closing volumes.[40]

Patients with ARDS who require mechanical ventilation and PEEP therapy are often sedated and pharmacologically paralyzed in order to facilitate respiratory support. Decisions concerning the need for sedation and paralysis are usually based on general clinical criteria. There is now evidence, obtained from experiments on normal volunteers, that the beneficial effects of PEEP on lung volumes are compromised significantly by the hyperpnea that often accompanies the clinical syndromes requiring such support.[41] Thus, appropriate sedation and neuromuscular blockade should always be considered when planning the ventilatory management of patients requiring more than minimal PEEP therapy.

The critical PEEP level necessary for alveolar recruitment may vary depending upon several considerations including the degree of lung pathology, the region of lung with PEEP-responsive pathology, and the patient's state of consciousness and neuromuscular blockade.

Cardiovascular Effects of PEEP

The cardiovascular effects of PEEP are attributable to several mechanisms. The primary mechanisms are mechanical and include decreased venous return to the heart, right-ventricular (RV) dysfunction, and alterations in left-ventricular (LV) function.

Decreased Venous Return

PEEP increases the mean airway pressure and, consequently, the pleural, mediastinal, and pericardial pressures. It has been known for some time that this general increase in intrathoracic pressure results in increased impedance to systemic venous return and is a major factor in the reduction in cardiac output associated with PEEP.[42-44] It has also been demonstrated that preload augmentation can offset these effects, restore stroke volume, and normalize cardiac output.[43] More recent studies utilizing various new technologic methods have been applied to further investigate and confirm these data.

Magnetic resonance imaging has been used in normal volunteers to study the effects of positive pressure ventilation and the application of PEEP on the heart.[45] This study revealed important configurational changes of the heart in the chest and changes in the sizes of all cardiac chambers in association with positive pressure ventilation and PEEP therapy (Fig. 6). Atrial volumes progressively decreased with increased PEEP, as did ventricular end-diastolic volumes and stroke volumes. These findings are consistent with decreased venous return due to cardiac compression by the increasingly expanded lungs. It is unfortunate that the study design did not separate the effects of positive pressure ventilation and the titration of PEEP; therefore, a combination of factors may well be responsible for the findings noted.

Two-dimensional Doppler echocardiography has also been used to assess variables that help define the mechanisms by which cardiac function is affected by PEEP therapy. Incremental titration of PEEP from 0 to 12.5 cm H_2O revealed a progressive decrease in end-diastolic volume index and cardiac index, but other indicators of ventricular function remained normal.[46] This provides further substantiation that decreased venous return is the primary factor responsible for decreased cardiac function induced by PEEP levels < 15 cm H_2O.

Cardiac function during PEEP has also been assessed with pulmonary artery catheterization employing advanced thermodilution cardiac output methods. Such methods utilize rapid-response thermistors that permit the assessment of beat-to-beat changes in intracardiac temperature and allow the calculation of RV ejection fraction and other variables during cardiac output determinations. Studies utilizing this technology have demonstrated variable results; however, a decrease in RV end-diastolic volume (RVEDV) consistent with decreased ventricular preload has been noted during titration of PEEP from 0 to 15 cm H_2O.[47,48]

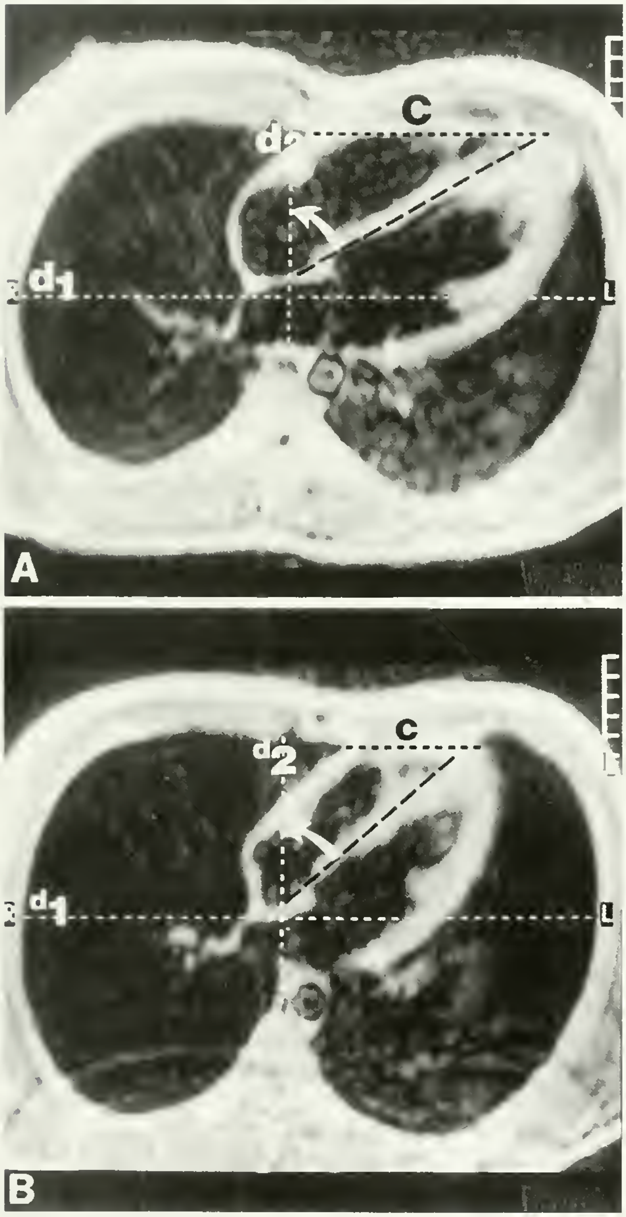

Fig. 6. Magnetic resonance images of the chest. The scan was taken at mid-diastole during spontaneous ventilation (top) and during positive pressure ventilation (bottom). The transverse-oblique chest diameter (d_1) remained nearly unchanged. There are marked changes in the sagittal-oblique chest diameter (d_2), in the length of contact between the heart and the anterior chest wall (c), and in the angle between the sagittal plane and the interventricular septum (curved arrow). (Reproduced, with permission, from Reference 45.)

Right-Ventricular Dysfunction

Many studies,[47-50] regardless of the methods used, demonstrate no clinically important impairment of RV function with PEEP levels < 15 cm H_2O. With higher levels of PEEP, especially when applied to normal lungs, there is evidence that RV afterload increases significantly and causes increases in RVEDV with a subsequent reduction in stroke volume.[49,50] These observations have been further substantiated by the advanced thermodilution cardiac output methods.[48] Figure 7 demonstrates the relationship between RV peak-systolic pressure and RV end-systolic volume index (RVESVI) as PEEP is increased from 0 to 20 cm H_2O. RV function is not impaired until the PEEP level exceeds 15 cm H_2O; but at 20 cm H_2O of PEEP, RV function begins to fall. However, in patients with underlying RV dysfunction, even small amounts of PEEP have the potential to cause important changes in RVEDV and RVEF.[51]

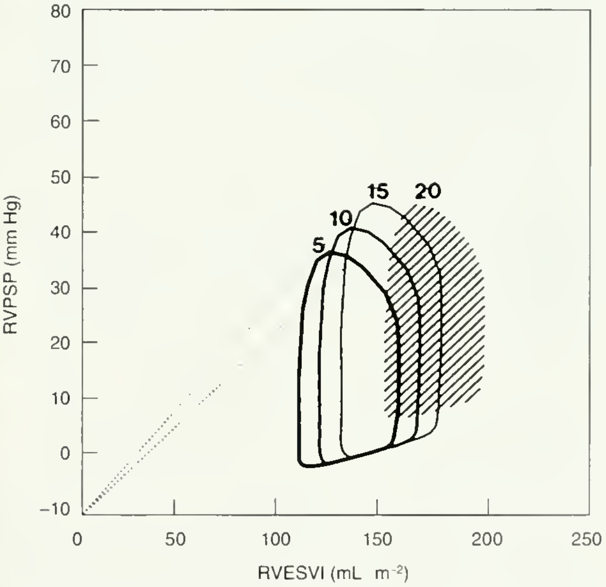

Fig. 7. Plots of right ventricular peak-systolic pressure (RVPSP) versus right ventricular end-systolic volume index (RVESVI) during titration of positive end-expiratory pressure (PEEP) levels. RVPSP increases linearly with increases in RVESVI as PEEP is increased up to 15 cm H_2O, thus, indicating no change in contractility. At PEEP levels > 15 cm H_2O, the slope of the RVPSP-RVESVI curve falls indicating a decrease in contractility. (Reproduced, with permission, from Reference 47.)

Altered Left-Ventricular Function

In addition to effects on venous return and RV function, evidence suggests that PEEP also has important effects on LV function.[52] The application of PEEP to the lungs increases RV afterload due to increased pulmonary vascular resistance. This results in RV dilation, which serves to maintain a constant stroke volume. This RV dilation alters the geometric configuration of the heart and results in a leftward displacement of the intraventricular septum. The septal shift encroaches upon the LV end-diastolic capacity and results in a diminished LV stroke volume. Recent combined thermodilution and echocardiographic studies have substantiated earlier findings.[53] One study[47] using two-dimensional Doppler echocardiography has failed to demonstrate this RV and LV interaction; however, this may be a function of the level of PEEP (< 15 cm H_2O) used in the study. Intravascular volume expansion can restore normal LV stroke volume by re-establishing normal LV end-diastolic capacity.[54]

Decreased distensibility of the left ventricle during PEEP therapy also appears to be directly related to the transmission of elevated pressures to the heart from the lung. Evidence for this has been generated with magnetic resonance imaging, which demonstrated extrinsic cardiac compression with the application of PEEP to normal lungs.[46] In addition, animal studies have shown that physically separating the lungs from the heart abolishes the adverse effects of PEEP therapy on LV end-diastolic volume.[50] Thus, the major mechanisms responsible for adverse alterations in LV distensibility appear to be ventricular septal shifting and cardiac compression.

In patients with underlying LV dysfunction and elevated filling pressures, PEEP may improve cardiac function by improving coronary arterial oxygen content and augmenting systolic contraction or by reducing venous return.[55] The reduction in venous return may actually produce a salutary effect by shifting the end-diastolic volumes to a position on the Starling curve associated with improved myocardial function.

Other Effects of PEEP

Possible Humoral & Established Anatomic Effects

Several other cardiovascular effects of PEEP have been reported. PEEP applied to one of two cross-circulated dogs resulted in reduction of cardiac output in both animals.[56] This and similar evidence has led to the hypothesis that negative inotropic humoral factors are released by the lung when PEEP is applied.[57,58] Precisely what these humoral factors might be is yet to be determined.

The lack of response (improvement in oxygenation or $\dot{Q}_{sp}/\dot{Q}_{tot}$) may indicate lung pathology not responsive to PEEP, but it may also indicate adverse cardiac effects of PEEP. It has been demonstrated, using contrast transesophageal echocardiography, that patients with a patent foramen ovale (PFO) may not respond to PEEP therapy because of increased anatomic right-to-left shunting with the application of PEEP to the lungs.[59] In light of the high incidence of PFO in the general population (20-34%),[60] this phenomenon should be considered whenever PEEP therapy does not produce the expected response. The cardiopulmonary effects of PEEP are known to occur within several minutes; however, precisely how long it is necessary to wait to measure the variables of concern following a PEEP change had not been documented until recently. It has now been shown that the greatest change in cardiopulmonary function occurs within the first minute following a change in PEEP level and that no further changes in oxygen delivery occur after 15 minutes.[61] This suggests that reassessments of cardiopulmonary function following PEEP changes should be made at 15 minutes.

Endocrinologic Effects

It has been known for some time that positive pressure ventilation and PEEP therapy induce an antidiuresis and antinatriuresis.[62] The mechanism by which this occurs has been investigated in various ways. The etiology of these effects appears to be multifactorial, involving both hemodynamic perturbations (reduction in preload and cardiac output) and endocrinologic processes.

The neuroendocrine processes that affect renal function involve several hormonal systems. The sympathetic nervous system affects renal function through regulation of the renin-angiotensin-aldosterone pathway. Other hormones that regulate water and electrolyte metabolism include arginine vasopressin, or AVP (also known as antidiuretic hormone, or ADH), atrial natriuretic peptide (ANP), and brain natriuretic peptide (BNP). All of these neuroendocrine pathways interact to maintain fluid and electrolyte homeostasis. The application of PEEP alters these pathways at various points and results in perturbations of fluid and electrolyte balance.

High sympathetic tone resulting in increased plasma renin activity (PRA) appears to be a major factor in the sodium and water retention noted with positive pressure ventilation and PEEP therapy.[63] The increased PRA activates the renin-angiotensin-aldosterone cascade and results in antinatriuresis and antidiuresis. There is preliminary evidence that increased renal prostaglandin synthesis tends to offset renal vasoconstriction and counterbalance the antidiuretic and antinatriuretic effects of increased sympathetic tone; however, the level of prostaglandin increase does not appear sufficient to completely counteract the effects in question.[64]

ADH is an important factor in the regulation of water metabolism. The primary determinant of ADH release is plasma osmolality; however, there are many nonosmotic factors that influence ADH release as well. These factors include blood pressure, nausea, vomiting, and various drugs (eg, narcotics and nonsteroidal anti-inflammatory agents). The cardiovascular effects of PEEP therapy tend to activate the hemodynamic regulatory mechanisms of ADH release.[64] This lowers the threshold for ADH release at any given plasma osmolality, increases ADH secretion, and results in water retention.

ANP and BNP play an important role in the regulation of water and sodium balance. Both of these substances promote diuresis and natriuresis. ANP is produced in and released from atrial myocytes; release is stimulated primarily by atrial stretching.[65,66] BNP was originally isolated from porcine brain; however, it is produced and released primarily from the ventricle of the heart.[67] Application of PEEP to postoperative patients has been demonstrated to decrease the levels of ANP and BNP and the levels of cyclic guanosine monophosphate, the secondary messenger of these hormones. Stepwise increases in PEEP applied to patients in acute respiratory failure have demonstrated a decrease in ANP levels when PEEP levels reach and exceed 10 cm H_2O.[68] In light of the facts that PEEP has been demonstrated to result in a decrease in cardiac size and that these hormones are released in response to distention of the cardiac chambers, it is logical that PEEP therapy would affect these hormonal systems in this fashion and result in antinatriuresis and antidiuresis.

PEEP Management Strategies: Low vs High PEEP

One must consider two aspects when discussing PEEP management strategies: (1) the definition of high versus low PEEP and (2) the determination of differences in outcome. The differentiation between high and low PEEP is controversial and variable depending upon the clinical situation. In addition, the definition and measurement of outcome present many debatable options.

In the neonatal population, PEEP levels of 3-6 cm H_2O have been demonstrated to consistently improve oxygenation without impairment of carbon dioxide excretion or hemodynamic stability.[69] However, other studies in this same patient population have looked at changes in tidal volume and carbon dioxide excretion as outcome end points and have indicated that PEEP levels of 2.5-3.5 cm H_2O may be preferable to PEEP levels of 4-6 cm H_2O.[70] A prospective, randomized study in newborn infants requiring extracorporeal membrane oxygenation (ECMO) due to respiratory failure looked at the effects of low (3-5 cm H_2O) versus high (12-14 cm H_2O) PEEP levels on lung function and recovery time.[71] The authors demonstrated that high PEEP levels prevented deterioration in lung function during ECMO and resulted in more rapid recovery and discontinuation of ECMO support than the use of low PEEP levels (mean [SD] for high PEEP: 97.4 [36.3] hours, low PEEP: 131.8 [54.5] hours; $p < 0.01$). It is studies such as this that provide the most pertinent information in this debate.

PEEP management strategies have not been well studied in the ARDS patient population. Although it is clear that PEEP therapy during ARDS impacts oxygenation and lung function in a positive fashion, randomized studies with well-defined outcome variables have not been undertaken, to date. Two retrospective studies have attempted to address the effects of high PEEP levels (> 15 cm H_2O) on physiologic function, complications, and mortality in ARDS.[72,73] Both of these studies were published by the same group and utilized a consistent management style in which PEEP was titrated to maintain a $\dot{Q}_{sp}/\dot{Q}_{tot} < 0.2$ and an $F_{IO_2} < 0.5$. They compared mortality rate with historical controls and concluded that survival was improved in their high PEEP population (PEEP range 15-50 cm H_2O). In addition, they found that the barotrauma noted was mostly related to initial traumatic injuries and that appropriate attention to hemodynamic monitoring and support resulted in minimal PEEP effects on cardiovascular status. The authors indicate that they are currently involved in a prospective, multicenter, randomized trial of different PEEP management strategies to further determine the role of PEEP in patient outcome and resource utilization.

The level of PEEP that should be considered low versus high should not be based upon arbitrary assignment of numeric values; rather, this concept should be based upon the physiologic effects that the therapy produces. PEEP levels that primarily result in increases in alveolar size and carry little in the way of hemodynamic effects should be considered low PEEP. In the adult patient population, this would be PEEP levels of up to 15 cm H_2O (Fig. 7).[47] PEEP levels that result in a significant amount of alveolar recruitment and that affect hemodynamic function should be considered high PEEP. The absolute values as applied to the pediatric population are less well defined because of the variability in patient size; however, the concept remains valid and applicable.

In Conclusion

A great deal of controversy and conflicting data exist pertaining to both the positive and negative effects of PEEP therapy. Reasons for this include such things as variations in the responses of animal versus human subjects to therapeutic interventions, differences in the effects of PEEP in normal and pathologic states, and methodologic and technologic variances among studies. It is important to remember these and to interpret data carefully and with as much consideration of these various factors as possible. In addition, the application of PEEP both to normal and pathologic lungs results in a complex set of events that impacts the entire organism, not simply the lungs or the oxygenation status. Failure to take this into consideration results in failure to provide optimal care for those patients who require it the most.

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Peruzzi Discussion

Kacmarek: Bill, a question about that Miller, Nelson et al study:[1] I'm not familiar with that particular study. You indicated that at PEEP levels greater than 15 cm H_2O there was a higher mortality. What was the difference in the mortality between the two groups?

Peruzzi: Actually, they did a retrospective review of their patients and chose those who required more than 15 cm H_2O PEEP—the level that they considered to be high PEEP. They looked at the mortality of those patients compared with the historical mortality of ARDS and found that when they were using more than 15 cm H_2O PEEP, and they were able to keep the shunt fraction less than 20%, mortality was only about 20%, which they interpreted as a significant improvement over historical mortality. They did this in preparation for a prospective, randomized study, which I have not seen as yet. They did not really have a control group, per se, in that study.

Hess: That group also published this month in *Critical Care Medicine*[1] and showed similar results in another retrospective study, which, quite honestly,

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[1] Miller RS, Nelson LD, DiRusso SM, Rutherford EJ, Safcsak K, Morris JA Jr. High level positive end-expiratory pressure management in trauma-associated adult respiratory distress syndrome. J Trauma 1992;33(2):284-291.

I find rather bothersome. In both studies[2] they ventilated patients with very high airway pressures—higher airway pressures than many of us in this room would be comfortable using. I am interested in hearing what some of the thoughts of other people around the room are, but one of my thoughts is that they're dealing with a different type of ARDS patient than perhaps many of us see in the ICU. Many of their patients are trauma patients. They're relatively young and previously healthy, and I think they're different from the kind of ARDS patient that we see in a medical ICU—patients who are septic and so forth. I'd be interested in the thoughts of others around the room, but I find that these studies bother me. I don't know quite what it is that bothers me about them, but their approach to PEEP is different from ours. They are essentially using gas-exchange targets and driving PEEP to high levels—20 cm H_2O or higher to achieve a targeted level of shunt. They try to get the shunts as low as they can. That leads me to a question: in your practice, assuming that hemodynamics are acceptable, do you titrate PEEP according to gas-exchange targets, or mechanics targets, or both? What is the role of the lower inflection point (P_{flex}) in this approach and so forth?

1. Di Russo SM, Nelson LD, Safesak K, Miller RS. Survival in patients with severe adult respiratory distress syndrome treated with high-level positive end-expiratory pressure. Crit Care Med 1995;23(9):1485-1496.
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Peruzzi: We tend to use the least-PEEP strategy, in that we try to use as much as necessary to maintain adequate oxygenation at what we consider to be subtoxic oxygen levels. (We do not specifically titrate PEEP based on lung mechanics except in certain circumstances.) This involves two issues, one of which is to be covered later on in this conference—modes of mechanical ventilation in ARDS and the best mode for using PEEP. We also use PEEP conservatively. However, what we have changed is how we approach ventilating ARDS patients. Once they begin to show the mechanics associated with alveolar overdistention, high plateau pressures as you've described, we tend to shift to a pressure-control mode of ventilation and try to alleviate some of the alveolar overdistention associated with tidal ventilation—but still use an appropriate level of PEEP to maintain adequate oxygenation. We accept a level of hypercapnia in those patients. From what you've described I do not think that Miller's group is doing that, and that obviously is a difference in philosophy. But we tend to titrate PEEP basically to oxygenation end points, and then when we get to end points involving lung mechanics, we start to look at different modes of mechanical ventilation.

Hess: I was going to call on Art (Slutsky) to say a little bit about some of his work with opening and closing of alveoli and the effect of shear forces.

Slutsky: In fact, Dean, I was going to ask a question about that. Bill, you didn't mention anything about the potential beneficial effects of PEEP, not related to gas exchange. There are data in the literature,[1-4] which you hinted at, suggesting that PEEP decreases the opening and closing of lung units and hence decreases the stress forces that can injure the lung. After reviewing the literature, what are your thoughts about this issue?

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4. Muscedere JG, Mullen JB, Gan K, Slutsky AS. Tidal ventilation at low airway pressures can augment lung injury. Am J Respir Crit Care Med 1994;149(5):1327-1334.

Peruzzi: The literature that I have reviewed suggests that PEEP application reduces the degree of alveolar recruitment associated with tidal ventilation. The animal data certainly suggest that avoidance of such alveolar stress factors may improve lung function and, potentially, outcome; however, I am aware of no human studies that have addressed the clinical outcomes associated with that. There is a lot of literature associated with PEEP in COPD and the effects on airway resistance. That really wasn't the focus of what I was trying to present because that's a different concept. I think that there's a great deal of interesting literature on that topic, and there, indeed, appear to be beneficial effects of PEEP in terms of closing volumes. We've gone from a circumstance where it was contraindicated to use PEEP in patients with reactive airway processes to the point now where we're considering it in certain patients in whom we think it may actually improve distribution of ventilation. However, I think a good number of the controversies in ARDS relate to choices of modes of mechanical ventilation, not just PEEP levels.

Slutsky: Dean, in response to your previous question: this issue is covered in my talk, which is next, because it has implications for high-frequency ventilation. In fact, I think if high-frequency ventilation is beneficial, it probably relates to the fact that the end-expiratory pressures are kept as high as possible, minimizing opening and closing of alveolar units, while also minimizing end-inspiratory lung stretch. I'll show you some animal data during my talk suggesting that PEEP is beneficial. Unfortunately human data are lacking. I didn't see the study this month by Miller, Nelson, et al[1] but the concept of minimizing the opening and closing of lung units might provide a rationale for using very high PEEP levels. What you want to do is recruit the lung. There are a couple of ways to know whether the lung is recruited. One way is to look at the pressure-volume curve, and I'll show some data using that approach, but the other way is to look at gas exchange. If your P_{O_2} increases, you're likely recruiting more lung units, and that may be the rationale underlying those studies.

1. DiRusso SM, Nelson LD, Safcsak K, Miller RS. Survival in patients with severe adult respiratory distress syndrome treated with high-level positive end-expiratory pressure. Crit Care Med 1995;23(9):1485-1496.

Hess: I guess the only problem with that is that when you drive the PEEP up to 20-25 cm H_2O, then you're left either with driving the airway pressures up to very high levels and getting into the risk of ventilator-induced lung injury, and all the Dreyfuss rat models sort of stuff, or you're limited to ventilating at very low tidal volumes and allowing very high P_{CO_2} levels. I guess the thing that troubled me about the Miller-Nelson study was that they used high levels of PEEP, and they allowed the airway pressures to be very high—55-60 cm H_2O, or so. Something about all of that just doesn't quite sit right, I guess. I'm bothered with their results, given the high airway pressures that they used, and the only explanation I can find is that these patients were ventilated for very short periods of time. These were, by and large I think, trauma patients who were relatively healthy previously and their ventilatory course was relatively short.

Slutsky: Was this a prospective study?

Hess: No, this was a second retrospective study. This was a follow-up study to the reference that Bill (Peruzzi) talked about. It incorporated more non-trauma-related ARDS patients and essentially came to the same conclusions as the first study. The prospective study is reportedly multicentered.

Slutsky: Did they have a control group as well?

Hess: I believe they used historical controls.

East: I just wanted to make a comment. I have talked to Loren (Nelson) about their study over the years, and I think their ARDS patients are just as sick as anybody else's. I think that it's not unique to that center, even. For instance, if you visit Harborview with Dave Pierson and Len Hudson, it's fascinating, because in the medical ICU, you'll find they use small tidal volumes and are very focused on keeping plateau pressures low. Yet, right next door in the surgical ICU, you have someone who trained with Civetta (Joseph), and they're using "super"-PEEP. I guess my whole take-home message from this paper and discussion is, maybe we don't know as much about it as we think we do, if people can use super-PEEP and get decent survivals.

Branson: We are involved in a multi-institutional trial that Loren Nelson is spearheading. The trial compares a high PEEP-low F_{IO_2} strategy to a low PEEP-high F_{IO_2} strategy. The goal of the study is to determine the relative risks of high PEEP to high F_{IO_2}. I think the issue of high airway pressures is not only "How high?" but also "In which patients?" The current acceptance of a plateau pressure of 35 cm H_2O is based on compelling evidence. However, isn't the issue transalveolar pressure? In surgical patients who have received 10 liters of crystalloid and packed red blood cells in the operating room, I believe chest-wall compliance is significantly reduced. I would guess that if we measured transalveolar pressure via pleural catheters or esophageal balloons, we would find that even with a PEEP of 20-25 cm H_2O and a PIP of 60 cm H_2O, the transalveolar pressure would be in the range that we consider safe. This is why pulmonologists are often shocked at the levels of support used by surgeons and surprised by the seemingly positive outcomes. Neil will talk about this issue tomorrow.

Haas: For the bedside practitioner, I have a question about performing "best"-PEEP studies. Do you monitor mixed venous saturation or do you measure cardiac output and calculate oxygen delivery? I'm curious about what others in this group use.

Peruzzi: We use both $S_{\bar{v}O_2}$ and continuous cardiac output monitoring, actually. We've become very enamored of the combination continuous cardiac output-$S_{\bar{v}O_2}$ monitor because it gives us a check and balance system. When cardiac output is dropping, we decide whether to do anything about it based on whether $S_{\bar{v}O_2}$ is also dropping, and vice versa. We find that it's very useful to use this balanced system to detect errors in measurement and to help make clinical decisions.

Adams: If there is a risk of crossing the lower inflection point of the P-V curve, then I would think that oxygenation shouldn't be the target you use to set PEEP because you could be just at that lower inflection point and crossing it with each breath. Is the creation of a P-V curve and determining exactly where that inflection point is important enough to take off the PEEP, derecruit and assess it properly, and then set the PEEP right above it?

Peruzzi: I think that is an interesting approach and a very good point. When one titrates PEEP to an end point, one oftentimes sees a rapid increase in oxygenation in association with that level of PEEP, and I think that the inflection point probably correlates with the change in oxygenation level. So, yes it is a reasonable thing to do if you're looking for alveolar recruitment, but I guess it's difficult to say that you can separate the two. I think that they're very closely related, and you're probably just looking at apples versus oranges; both of those happen to be doing the same thing at the same time.

I have not compared changes in pressure-volume curves with oxygenation changes to tell you whether, indeed, they correlate and if that would make a difference in my decisions.

Slutsky: I would just like to emphasize Rich's (Branson) point about the interpretation of airway pressures. Although we usually think of high airway pressures as predisposing patients to barotrauma, it is the transpulmonary pressure that is likely the more important pressure in this regard. Thus, if the patient has a very stiff chest wall as occurs with massive ascites, a high airway—or even a high alveolar pressure—may not be really of major concern because pleural pressure will also be elevated. This doesn't mean that you have to put an esophageal balloon into everybody, but consider it an important point to keep in mind. The second point that I'd like to make is that if you look at the animal models, all it takes is a couple of hours, or less, of inappropriate ventilation to cause the kind of structural damage that could take weeks to heal. So, it's important to apply the correct ventilatory strategy very early on, before the ventilator actually damages the lung.

Hess: I think we have to wait to see the end of the randomized trial.

Peruzzi: The other point that I would make is that it's clear to me from my experience, especially in dealing with acute lung injury and oxygen toxicity, that humans and animals are very different. Animals appear to be much more sensitive to these sorts of problems than humans, and it's a question that is going to require a randomized and prospective study to answer.

Hess: Since Bob is the summarizer, he gets the last point.

Kacmarek: I wanted to comment on Alex's (Adams) point. I agree that the use of inflection points to set PEEP is a nice theoretical approach. My experience is that at the bedside it's not the easiest thing to do, nor do you see an inflection point in all patients. I personally find that in the majority of patients, unless they're young, previously healthy, trauma victims without pre-existing lung disease, it's very difficult to identify an inflection point. Even if you go to a square wave pattern, with long, protracted inspiration or do multiple inflations with a giant syringe. It's just something that you can't always identify clinically.

Peruzzi: I think that also goes along with what Gattinoni showed,[1] that in certain lung areas and in certain patients, the pathology isn't going to result in alveolar recruitment, to a significant degree. You're going to increase alveolar size, you'll redistribute water, but you won't necessarily recruit a phenomenal number of alveoli. Whether that's going to be a useful technique depends on what the pathology is in that individual and the extent of that pathology.

1. Gattinoni L, D'Andrea L, Pelosi P, Vitale G, Pesenti A, Fumagalli R. Regional effects and mechanism of positive end-expiratory pressure in early adult respiratory distress syndrome. JAMA 1993;269(16):2122-2127. *See the published erratum JAMA 1993;270(15):1814.*

Hess: The point that I was going to make is that, really, you're interested in the inflection points of the recruitable alveoli. When you look at something here at the mouth, you're looking overall at what's happening.

Branson: This will be very helpful, and I'm going to put Alex on the spot, since he's drawing pictures. I am asked repeatedly, "How do you measure the inflection point in the ICU?" and for readers of the Journal, if you would just take a few seconds to tell us how to set the flow pattern, and other ventilator variables—or is it better to use a super syringe? Do you have to have esophageal pressure or can you just do it with the ventilator? All those things would be very, very helpful to the people who take care of patients at the bedside all the time.

Adams: I certainly can't answer that. I can cite the two studies[1,2] that have reduced PEEP in patients and used the plateau pressures established by incremental increases in tidal volume, looking for a line, and then trying to establish some criterion for where the curve at the bottom and the curve at the top leave that line. There could be sophisticated curve fittings that could find it, but in both cases, as was pointed out by the presenter, these are regions that aren't that distinct. But, is it risky to be that far above it if you make a mistake? It may be risky to be in it, and that's what I would want to know. I'm faced with the same questions that you ask.

1. Roupie E, Dambrosio M, Servillo G, Mentec H, El Atrous S, Beydon L, et al. Titration of tidal volume and induced hypercapnia in acute respiratory distress syndrome. Am J Respir Crit Care Med 1995;152(1):121-128.
2. Amato MBP, Barbas CSV, Medeiros DM, Schettino GdePP, Filho GL, Kairalla RA, et al. Beneficial effects of the "open lung approach" with low distending pressures in acute respiratory distress syndrome: a prospective randomized study on mechanical ventilation. Am J Respir Crit Care Med 1995;152(6):1835-1846.

Tracheal Gas Insufflation (TGI)

Alexander B Adams MPH RRT

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Introduction

> At the end of expiration, dead space between the carina and the external orifices of the airways mainly contains air with the composition of alveolar air; could this part of the dead space be eliminated, tidal volume would be decreased by a corresponding volume. Furthermore, CO_2 normally contained in this part of the airways no longer would be rebreathed, leading to a decrease in minute ventilation as a physiological response set to maintain the existing CO_2 level.[1]

This 1969 communication from Severinghaus to Stresemann[1] specifically and accurately describes the primary effect of tracheal gas insufflation (TGI), that is, the flushing of anatomic and apparatus dead space. A current definition for the adjunctive role of TGI in mechanical ventilation in a pressure-guarding ventilatory strategy is the continuous or phasic injection of fresh gas into the central airways for the purpose of improving the efficiency of alveolar ventilation and/or minimizing the ventilatory pressure requirement.

In this paper, TGI is discussed, primarily, as an adjunct to mechanical ventilation. Two other variants of TGI have been extensively investigated and implemented and are discussed briefly. Transtracheal oxygen (TTO) refers to TGI administered to a spontaneously breathing subject and has been used since the early 1980s.[2,3] TTO provides a means of oxygen delivery that allows reduced oxygen flow to provide equivalent oxygen saturation. This reduces oxygen costs and extends the supply, which may be particularly important in the home setting. In addition, TTO has been shown to reduce minute ventilation by the mechanism described by Severinghaus.[4-6] Consequently, TTO reduces the work of breathing and improves exercise tolerance.[7,8] Finally, the catheter can be concealed for cosmetic purposes because it enters the trachea percutaneously above the sternal notch. The other TGI variant, continuous apneic ventilation, delivers gas to an apneic animal or patient and, at certain catheter positions and flows, provides adequate gas exchange. Variations of continuous apneic TGI have been termed constant-flow ventilation,[9] constant oxygen insufflation,[10] continuous-flow apneic ventilation,[11]

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A version of this paper was presented by Mr Adams during the RESPIRATORY CARE Journal Conference, Mechanical Ventilation: Ventilatory Techniques, Pharmacology, & Patient Management Strategies, held in Cancún, Mexico, October 6-8, 1995.

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and tracheal insufflation of oxygen.[12] Even though clinical indications are few, this variant of TGI has enlightened investigators to proper positioning, flows, and the mechanisms of action of TGI.[9-12]

Recently, TGI has been applied during mechanical ventilation to improve oxygenation. Although oxygenation in mechanically ventilated patients often improves with TGI, it should not be applied solely to achieve that goal. The placement of a catheter in the trachea of a ventilated patient is technically simple but functionally complex. A simple circuit for TGI is shown in Figure 1. Continuous TGI by this method presents complex questions about delivery efficacy, ventilator interaction problems, and potential complications.

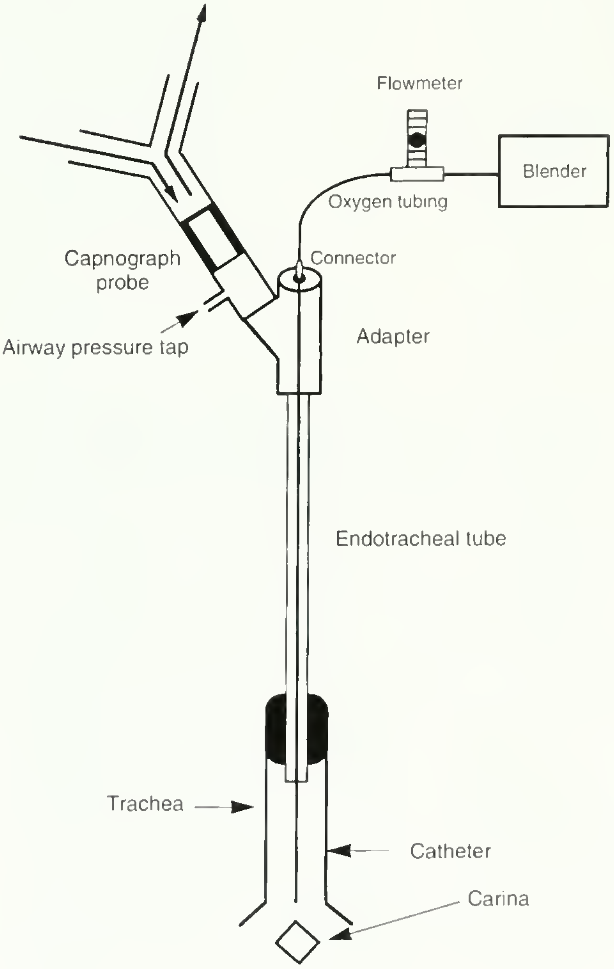

Fig. 1. A simple circuit for tracheal gas insufflation. The ventilator circuitry includes the Y adapter, a capnograph probe, an airway pressure tap, a bronchoscopy adapter, and the endotracheal tube. The tracheal gas delivery components include a blender to deliver a ventilator-matched oxygen concentration, a flowmeter to set the tracheal gas flow, oxygen tubing, a connector for the tubing-bronchoscope adapter, and the catheter guided through the bronchoscopy adapter to a position above the carina.

Technical Aspects of TGI

Catheter Placement & Shape

For the purpose of dead-space flushing, the flow (catheter tip) can be introduced in the region of the carina. The precise position of the tip of the catheter is, probably, less important than might be expected. The catheter can be placed above the carina under bronchoscopic guidance or placement can be estimated from a recent chest radiograph. In addition to simple dead-space flushing (proximal effect), a distal effect further improves TGI efficiency and is caused by a jet exiting the catheter. Beyond the jet is another region of enhanced turbulence (Fig. 2). The extent of this region of turbulence is related to flow velocity and is increased by increasing catheter flow and decreasing lumen size. The work of Slutsky and Menon[13] using an animal model revealed that advancing the catheter more than 3.5 cm beyond the carina greatly enhances gas mixing (and, therefore, the distal effect) but can also cause regional hyperinflation.[13] Although the distal effect enhances CO_2 extraction, the presence of the catheter and the jet effect oppose usual expiratory flow. The need to consider both proximal and distal effects has led to the development of novel approaches to directing the tracheal gas flow. Isabey et al[14] have designed a TGI endotracheal tube (ETT) that allows gas to be injected through capillaries imbedded in the tube, thus, avoiding circuit interruption to add tracheal gas and eliminating the catheter within the trachea. Kolobow and co-workers[15] have developed a reverse-thrust catheter that directs tra-

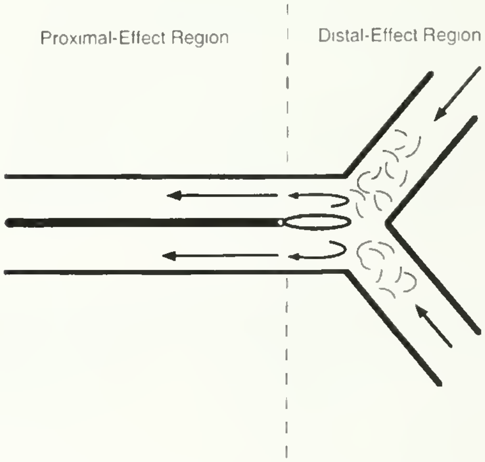

Fig. 2. Tracheal gas insufflation effects within the trachea and mainstem bronchi. The proximal effect is due to the dead-space flushing from the catheter tip to the Y of the ventilator circuit. The distal-effect region consists of a jet area extending from the catheter tip and a turbulent region extending beyond the jet towards the alveoli. The extent of the jet and turbulence regions is related to flow velocity at the catheter tip. The velocity is directly related to flowrate and inversely related to the internal diameter of the catheter.

cheal gas flow back towards the ventilator-circuit Y, thus, accomplishing expiratory flushing and avoiding hyperinflation. Burwen et al of Slutsky's group[16] have placed the TGI catheter along the trachea outside of the ETT, but the more standard approach is to place the catheter through the ETT.

Studies examining the shape of the catheter have confirmed that two effects can be anticipated from TGI. A catheter shaped to direct flow towards the Y functions, primarily, by flushing dead space (proximal effect). Directing the catheter towards the distal airways (straight catheter) should produce both distal and proximal effects. Our group[17] studied a straight and an inverted catheter and demonstrated that, with relatively low flows of 2-6 L/min and with the catheter 1 cm above the carina, 25% of the total reduction in P_{aCO_2} was due to distal effect and 75% was due to proximal effect (Fig. 3).

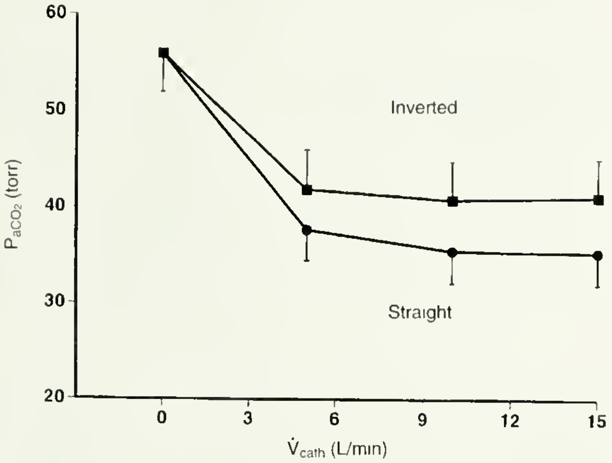

Fig. 3. The relationship between catheter flow and P_{aCO_2}. To quantify the proximal and distal tracheal gas insufflation effects in dogs, gas ($\dot{V}_{cath}$) was insufflated at 5, 10, and 15 L/min through a 1.6-mm internal diameter copper tube that had been bent to direct flow towards the Y (proximal effect) or was straight (proximal and distal effects). P_{aCO_2} was significantly less (means with error bars) than baseline at each flow for the straight and for the inverted tubes. The extra reduction seen with the straight tube represents the addition of distal effect.

Monitoring Effectiveness

The effectiveness of TGI overall is measured directly by P_{aCO_2} reduction. On a breath-by-breath basis, capnography displays the completeness of flushing. Although end-tidal CO_2 ($ETCO_2$) normally plateaus toward end-exhalation, TGI causes $ETCO_2$ to decrease with catheter flow and fall to baseline if all flushable CO_2 is removed (Fig. 4).[18] Unfortunately, capnography and TGI can have a somewhat complex relationship. The $ETCO_2$ only measures CO_2 at one place in the circuit at one time. For example, at the same catheter flow the straight catheter, because of distal flushing effects, may be delivering more CO_2 to the central airway and actually produce a higher $ETCO_2$ despite causing a lower arterial P_{aCO_2} than an inverted catheter that flushes only the proximal airway.

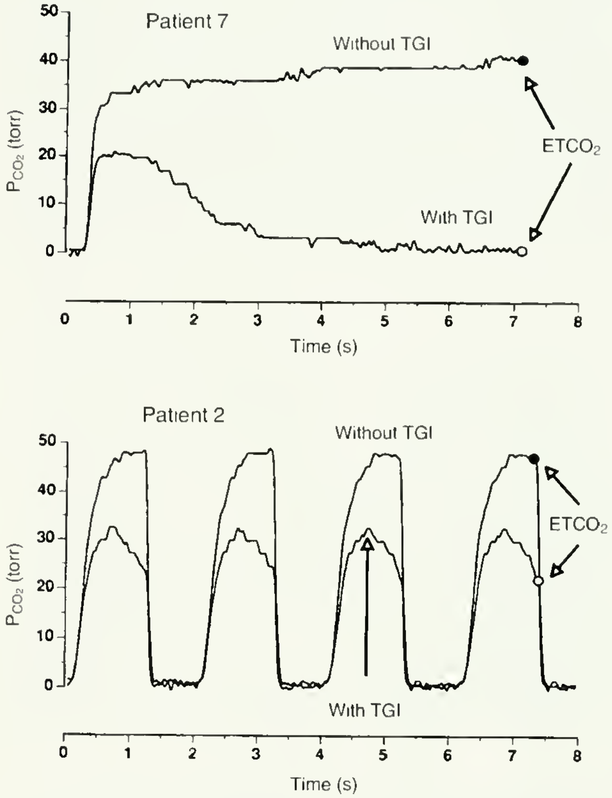

Fig. 4. Capnography tracings with and without tracheal gas insufflation (TGI). Representative capnography from 2 patients, each receiving usual and TGI-assisted mechanical ventilation. For Patient 7, the upper tracing was made without TGI while the lower tracing shows TGI returning the $ETCO_2$ to baseline by complete CO_2 flushing of the range of TGI effect. For Patient 2, the shortened expiratory time does not allow the capnogram to return to baseline (lower tracing) due to incomplete flushing. Note the differences in $ETCO_2$—the differences being directly related to P_{aCO_2} reduction due to TGI.

Ventilator-Cycle Phasing

Catheter Volume & Delivery

Tracheal gas can be delivered continuously, during all or part of expiration, or during inspiration only (inspiratory bypass—as a catheter route to deliver tidal volume).[19] Continuous TGI also delivers flow during inspiration that adds to the ventilator-delivered tidal volume and, thus, to minute ventilation. For example, with an inspiratory time of 1.5 seconds and a tracheal gas flow of 6 L/min, 150 mL is added to the tidal volume—a 25% increase in a tidal volume of

600 mL and a 25% increase in minute ventilation. This addition can be avoided simply by decreasing the set tidal volume accordingly or by utilizing expiratory TGI. Inspiratory-bypass TGI avoids the rebreathing of gas in the anatomic dead space but is the least effective of the three phasing options described.[19]

In Figure 3, P_{aCO_2} reduction by TGI is shown to be related to increased catheter flow. In a study of catheter flow and volume effects, our group[20] found that the volume of tracheal gas delivered during expiration is a major CO_2 extraction factor (Fig. 5). This expiratory volume must be delivered in part during the end-expiratory period to optimize efficacy. An equivalent volume delivered early in expiration is less effective.

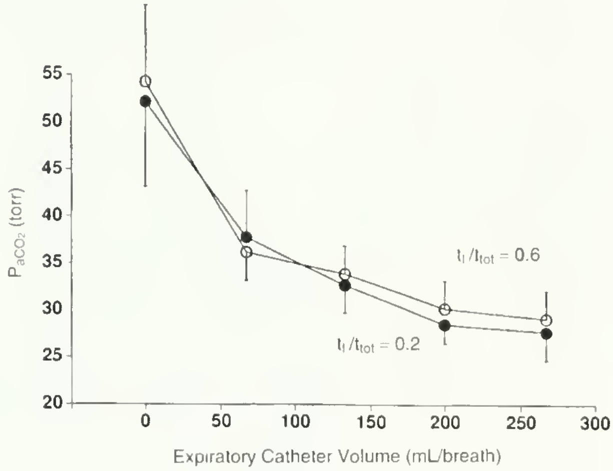

Fig. 5. Volume delivery of expiratory tracheal gas insufflation (TGI) and P_{aCO_2} reduction. To relate catheter-delivered volume to P_{aCO_2} reduction, mean P_{aCO_2} values are plotted for TGI catheter-delivered volume from several specific flow settings when the ratio of inspiratory time to total respiratory time, or t_I/t_{tot}, = 0.6 (open circles, ○) and t_I/t_{tot} = 0.2 (closed circles, •) In other words, low flows for longer periods were compared to high flows for shorter periods. Equivalent catheter volumes produced equivalent reductions in P_{aCO_2}; therefore, TGI flow and duration are important.

Vibration

An intriguing combination of therapies has been reported by Bitterman, Gavriely, and co-workers[21-23] in animal studies spanning more than a decade. They have found that the effects of TGI are enhanced by externally applied chest-wall vibration. This is logically explained as an expanded distal effect by vibration-increased turbulence (which might also be seen by vibrating or pulsing the insufflated gas).

Ventilator Gating of TGI: Technical Aspects

It appears that gas delivery during a certain phase of expiration is required to precisely control TGI and produce the desired effect. The question is How is the tracheal gas to be controlled? In 1968, Stresemann et al[24] sensed the phases of respiration and delivered tracheal gas solely during expiration. Modern mechanical ventilators both control and sense respiratory cycling through pressure and flow transducers. The flow and pressure signals are, usually, externalized and available for display, analysis, or as a potential gating mechanism for tracheal gas flow—simply put, a signal from the ventilator indicating positive flow can be set to halt tracheal gas flow to the patient during inspiration. The pulsing of gas delivery during the portion of expiration that maximizes CO_2 extraction and minimizes the effect on triggering is more complex. In an experimental setting, it has been possible to capture that signal and gate a solenoid, directing flow of insufflated gas during desired portions of expiration.[20] Continuing development of flow signal analysis and TGI control technologies are required to maximize TGI effectiveness and reduce the possibility of complications. If TGI is found to be more effective during a certain range of expiratory flow, that flow range could be assessed from the expiratory flow signal and could gate gas flow accordingly.

TGI Sabotages Ventilator Monitoring

Several important aspects of the normal function of a mechanical ventilator are altered by TGI. If these alterations are not recognized and accounted for, I believe the indiscriminate use of TGI with mechanical ventilation can be hazardous.

As mentioned earlier, continuous TGI adds tidal volume to the ventilator-delivered breath. Less well-known is that end-expiratory TGI can also add to tidal volume (Fig. 6).[19] In this situation, the patient's expiratory flow has ended but TGI continues. Tracheal gas usually exits the expiratory circuit, but when flow resistance toward the alveoli is less than the resistance of the expiratory circuitry and valve resistance, flow goes to the patient and adds to the subsequent ventilator tidal volume.

The introduction of a TGI bias flow into the circuitry, for example, has two apparent adverse effects. First, the tracheal gas flow impairs the accuracy of the expiratory pneumotachometer. TGI adds to the expired volume, which would not have affected many previous-generation ventilators; however, current ventilators evaluate the exhaled breath and can alarm if there is a mismatch between delivered and exhaled breaths. Of even greater importance is the inability of the expiratory pneumotachometer to detect a leak or the absence of a pressurized breath when TGI appears to be delivering an adequate exhaled tidal volume. Second, the TGI bias flow can affect the patient's ability to trigger the ventilator.[25] For the patient on TGI to trigger a ventilator-supported breath, an inspiratory effort must outstrip the tracheal gas flow *and* any addition to functional residual capacity (FRC) resulting from TGI. Figure 7 shows a patient attempting breaths during TGI but receiving ventilator-supported breaths in only 5 of 11 attempts.

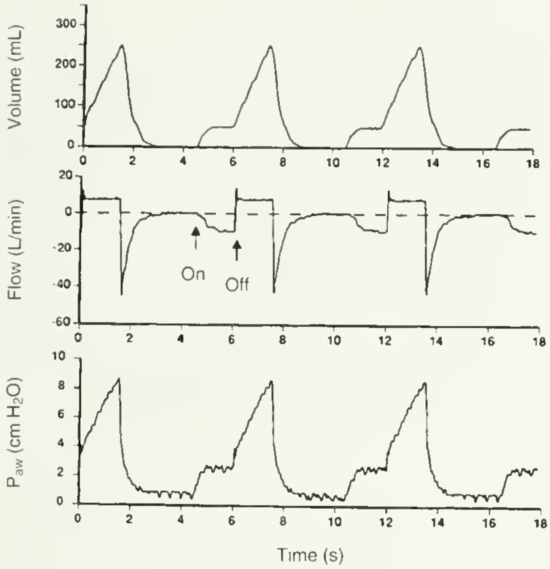

Fig. 6. The addition to inspiratory tidal volume by late expiratory gas insufflation (TGI). Volume, flow, and airway pressure tracings are shown for two complete breaths with late expiratory TGI. In this case, tracheal gas delivery begins during the ventilator expiratory phase. Because patient expiratory flow is complete when TGI begins, insufflated gas travels the path of lesser resistance (less resistance than the expiratory circuitry) to the lungs and adds to the next tidal volume before the ventilator cycles.

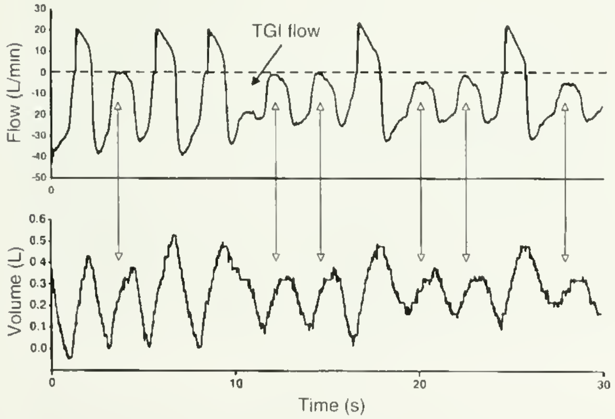

Fig. 7. Tracheal gas insufflation's preventing ventilator triggering. This patient received TGI of 10 L/min and could not trigger ventilator-supported breaths for 6 of the efforts (designated by arrows). Five breaths were supported by pressure support while the nonsupported breaths were shallower. Volume was measured by respiratory inductance plethysmography.

Of course the patient received smaller, unsupported breaths from the tracheal gas flow. In general, the extra effort needed to trigger breaths during TGI can be partially compensated for by designing a system of flow-triggering from the tracheal gas flow.

Continuous TGI also makes certain maneuvers hazardous if even possible. When either an end-inspiratory pause or auto-PEEP (ie, intrinsic positive end-expiratory pressure) is being evaluated, either the clinician or the ventilator must halt flow at end-inspiration or end-expiration. If TGI continues during the maneuver, a plateau or stable value can never be reached. Excess pressure exposure is then a risk.

The fractional concentration of oxygen delivered (F_{DO_2}) may be increased by TGI particularly with continuous TGI. If the insufflated gas has an FO_2 of 1.0, TGI modestly increases actual F_{DO_2} if the ventilator F_{DO_2} is set below 1.0. The lower the oxygen concentration set on the ventilator and the higher the tracheal gas flow, the higher the oxygen concentration received by the patient. This can be remedied by blending the TGI gas to match the ventilator-delivered concentration.

Another potential problem involves continuous TGI with pressure-control ventilation (PCV). In pressure-control ventilation, once the lung has filled to its resting level for the set pressure, continuous TGI continues to inflate the lungs and add to lung pressure (although the intent of PCV is to cap the pressure) until the end of the inspiratory period. This additional pressure has been evaluated in test lung studies.[26,27] This problem can be recognized by observation of the airway pressure tracing and is accentuated by long inspiratory times, lower compliance, and lower resistance. A pressure limit setting on the ventilator can be set to relieve the pressure or an additional valve can be inserted in the circuitry to vent the excess pressure.

Potential Complications

Continuous and pan-expiratory TGI elevate mean lung volume. This effect, in addition to increasing F_{DO_2} as previously described, can cause an increase in P_{aO_2} much as does PEEP. Because TGI opposes expiratory flow, it causes back pressure through the ETT and the expiratory valve. TGI can increase FRC as reported by our group; at flows of 2, 4, and 6 L/min, FRC was raised by an average of 25, 45, and 62 mL, respectively.[18] The auto-PEEP caused by TGI could become clinically important during inverse ratio ventilation, particularly with higher catheter flows.

Experience with TTO in spontaneously breathing patients is extensive. Compared to TTO patients, ventilated patients are more labile, have more secretions, and are probably more susceptible to the effects of jet shear to the tracheal wall and to so-called catheter whip (flipping motion of the catheter tip) than are spontaneously breathing patients. The infrequent serious consequences of the application of TTO can also occur with TGI. The formation of mucus balls and tracheal granulation have been associated with TTO[28-30]—probable consequences of drying. Our group[31] has shown that insufflated gas cools the central airways and that cooling is greater with continuous than with expiratory TGI (Fig.

8). This cooling can be lessened if the insufflated gas is heated and humidified, but the back pressure needed to drive the flow through the catheter exceeds the leak or burst pressure of most humidifiers. Figure 9 displays the back pressure needed to drive flow through catheters of 4 sizes. A pressure exceeding 110 cm H_2O causes leakage or bursting of most available humidifiers. Therefore, heating and humidifying gas for tracheal insufflation at higher flows through smaller catheters is a problem.

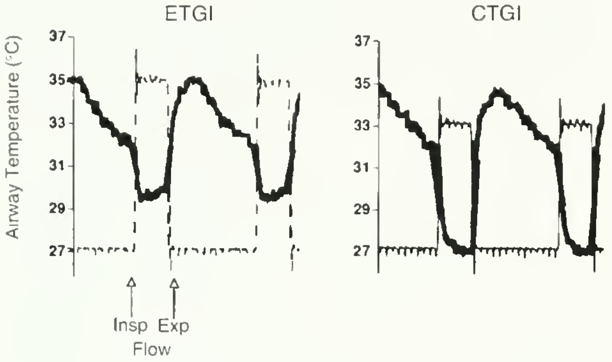

Fig. 8. Temperature declines in the central airway caused by tracheal gas insufflation (TGI). These are tracings from a thermistor measuring temperature in the central airways during TGI in a dog. Compared to baseline, both unheated expiratory TGI (ETGI) and unheated continuous TGI (CTGI) markedly cool the airways—CTGI to a greater extent than ETGI.

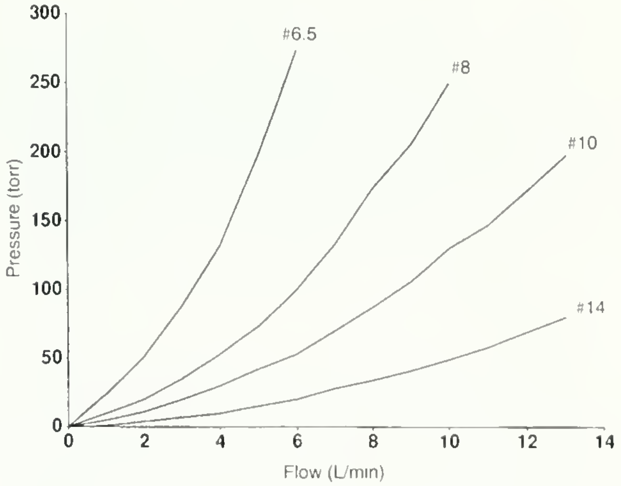

Fig. 9 Back pressure from flow through catheters of 4 commonly used sizes. High pressure is required to drive gas through catheters used for tracheal gas insufflation, with much higher pressures required for catheters with smaller lumens (6.5 Fr and 8 Fr). Because most humidifiers leak or burst at pressures above 110 torr, attempting to humidify gas for insufflation through catheters with smaller lumens may not be possible even at relatively low flows.

Clinical Studies

Clinical studies thoroughly evaluating TGI are lacking. Stresemann et al[24] first applied TGI to a ventilated patient in 1968. The patient was ventilated by a Bird Mark 8 while an inline thermistor detected expiratory flow and triggered a solenoid to insufflate gas to the trachea throughout expiration by a precision flowmeter. For this patient, TGI resulted in a reduction in P_{aCO_2} from 54 to 43.9 torr, and the mechanism of this fall was thought to be expiratory flushing. Not until the 1980s did investigators describe TGI in animal models as a method that would allow reduction in rate and tidal volume and, therefore, pressure exposure.[32,33] In 1991, Gilbert et al[34] described the use of expiratory TGI as part of a pressure-guarding strategy in a patient with ARDS. The patient had 11 chest tubes and a persistent 40% pneumothorax. The patient eventually recovered, but interpretation of the effect of TGI is difficult because the inspiratory-to-expiratory-time ratio of 4:1 (with auto-PEEP = 12 cm H_2O) was converted to 1:2 (auto-PEEP = 0 cm H_2O) when TGI was instituted. Our group[18] reported the first series of patients to whom TGI was administered—8 patients with a variety of underlying causes of respiratory failure. TGI reduced P_{aCO_2} in all patients, with an average reduction of 15%. In 1994, Nakos et al[35] reported the use of TGI in 7 patients with acute lung injury. Not only was P_{aCO_2} reduced by TGI but also a 24% reduction in tidal volume maintained an equivalent P_{aCO_2}. This application showed that the pressure-sparing effects proposed by earlier investigators could be used in the clinical setting.

Of the reported human studies, none have evaluated the role of TGI in permissive hypercapnia. TGI should be more effective by causing a greater reduction of P_{aCO_2} when P_{aCO_2} is elevated.[36] Studies of a role for TGI in ventilator weaning have not been published. Van Hooser et al[37] compared three forms of partial support with TTO only. That report showed that TGI was feasible in the marginally dependent patient, but the minute ventilation reducing effects of TGI were not and have not been studied in this patient group.

In Summary

- The proposed theoretical applications for TGI use are a pressure-guarding strategy and as a tactic in weaning the marginally dependent patient (based on studies using normal and injured animal models, investigating the technique, and TGI's effectiveness and safety).
- The effectiveness of TGI with the newer TGI-endotracheal tube designs, partial expiratory phasing, and TGI in conjunction with other therapies demand further study.
- Important safety issues remain regarding the TGI-ventilator interface and the catheter's effect within the patient.
- Clinical studies are few—particularly, studies of TGI in weaning and as an adjunct to controlling CO_2 extraction during permissive hypercapnia.

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Adams Discussion

Hess: A concern that I have is that with ARDS the dead space is alveolar dead space and not anatomic dead space. Our anecdotal experience with TGI hasn't been overwhelming. We have not been very impressed. Part of the explanation for that is—with ARDS you have this large alveolar dead space. TGI flushes out the anatomic dead space, but it doesn't—except for some of the distal effects that you talked about—affect the alveolar dead space that much.

Adams: Precisely. That same argument would also apply for chronic obstructive lung disease. In neuromuscular weakness, perhaps, where a larger portion of the dead space is anatomic, or in, perhaps, asthma, TGI can be expected to have a greater effect. However, as you point out, TGI would be expected to be less effective in acute lung injury and obstructive lung disease.

Wilson: I am interested in the data you presented looking at dead-space ventilation and improvement in P_{CO_2}. When Gattinoni et al[1] use this technique (and we'll discuss it a little bit more this afternoon when we talk about $ECCO_2R$), they use it for oxygenation and not for CO_2 removal. Could you comment on that?

1. Gattinoni L, D'Andrea L, Pelosi P, Vitale G, Pesenti A, Fumagalli R. Regional effects and mechanism of positive end-expiratory pressure in early adult respiratory distress syndrome. JAMA 1993;269(16):2122-2127. *See the published erratum JAMA 1993;270(15):1814.*

Adams: In the animal studies and human studies that our group[1,2] and others[3] have published, if the oxygen concentration delivered by TGI is the same as the oxygen concentration from the ventilator, and if mean thoracic volume is kept the same, there will be an increase in oxygenation due to TGI related to the P_{aCO_2} reduction. So, if one's intent is to apply TGI to increase oxygenation, one should consider other methods that are safer and more easily controlled.

1. Nahum A, Shapiro RS, Ravenscraft SA, Adams AB, Marini JJ. Efficacy of expiratory tracheal gas insufflation in a canine model of lung injury. Am J Respir Crit Care Med 1995;152(2):489-495.
2. Ravenscraft SA, Burke WC, Nahum A, Adams AB, Nakos G, Marcy TW, Marini JJ. Tracheal gas insufflation augments CO_2 clearance during mechanical ventilation. Am Rev Respir Dis 1993;148(2):345-351.
3. Nakos G, Zakinthinos S, Kotanidou A, Tsagaris H, Roussos C. Tracheal gas insufflation reduces the tidal volume while P_{aCO_2} is maintained constant. Intensive Care Med 1994;20:407-413.

Slutsky: I think that Gattinoni[1] uses tracheal insufflation sort of like apneic oxygenation as a source of oxygen to the lung, keeping the lung at a constant lung volume, so as not to cause further injury. So, it can certainly be used to improve oxygenation somewhat, but it's an unusual situation. Most of the gas exchange is taking place in the extracorporeal circuit. We've done some studies[2] that show that the oxygenation will be much greater when gas is delivered through a catheter in the distal airways rather than at the proximal airway (and, actually, you can get some CO_2 removal, as well, even during apnea).

1. Gattinoni L, D'Andrea L, Pelosi P, Vitale G, Pesenti A, Fumagalli R. Regional effects and mechanism of positive end-expiratory pressure in early adult respiratory distress syndrome. JAMA 1993;269(16):2122-2127. *See the published erratum JAMA 1993;270(15):1814.*
2. Long SE, Menon AS, Kato H, Goldstein RS, Slutsky AS. Constant oxygen insufflation (COI) in a ventilatory failure model. Am Rev Respir Dis 1988;138(3):630-635.

Wilson: The total gas flow that they use is targeted at the oxygen consumption of the patient, and that's how they titrate the gas flow—to replace that volume during the apneic period.

Kacmarek: The other thing you tend to fail to realize is that with TGI you're adding PEEP to the system. A constant flow at the tracheal level is just like turning up PEEP on the ventilator, and it's amazing how much you can generate in certain patients with 12-14 L/min, as some individuals are using. In lung models, we've noted up to 10 cm H_2O PEEP added to the system, with TGI flows at 12 L/min. I don't remember how much Gattinoni uses, but if you're above 10 L/min, you can create a lot of PEEP, which may be some of the explanation of the improvement in oxygenation. That's one of the things we saw clinically, when we first used it. More than seeing any effect on CO_2 removal, we saw tremendous improvement in oxygenation, which we could not explain until we went into the laboratory and really played with it and found that what we were doing was simply causing PEEP to be applied to the system.

Wilson: I think that you have a good point there, Bob. Auto-PEEP is not usually discussed with the $ECCO_2R$ technique. Gattinoni believes that the oxygenation in $ECCO_2R$ occurs by virtue of native lung, primarily, because it's a low-flow, by-pass technique. I really agree with you that the oxygenation improvement (and he's using low TGI flows, in the 1-5 L/min range), is really a function of the auto-PEEP that is created. The auto-PEEP may not be reported accurately in the $ECCO_2R$ studies that use TGI.

Branson: I guess, Alex, I don't have any further comment on that. The thing that I'd like to comment about is that when TGI first came out, it was touted as a fairly simple method of reducing P_{CO_2}. After your presentation I find that statement could not be farther from the truth. I think one of the things that Bob is pointing out is that the auto-PEEP issue is related not only to the flow but also to the size of the catheter you use and to the size of the endotracheal tube. With everything you've presented, where the catheter is, whether it has side holes or end holes, how big it is, how big it is in relationship to the endotracheal tube, how much you deliver in relationship to what the CO_2 is, and I guess from my clinical experience, TGI hasn't helped a whole lot in ARDS. To me, it's not a technique that is safe right now—in the average person's hands.

Adams: Correct, don't try this at home, folks! I was waiting for that comment because I agree completely. If it is to be used in the clinical setting in any manner for the reasons that you mentioned, a protocol should be established in which all of these precautions are known by those providing care—particularly, the ventilator management and how it can alter the ventilator interface.

Slutsky: That's in contrast to situations where alveolar dead space is low. We[1] did an experiment in which dogs were given small doses of paralytic agent, so their P_{CO_2} was maintained at about 90 torr. By using gas insufflation at a few liters per minute, the P_{CO_2} dropped from an average of 90 to about 65 torr, at the same time that the minute ventilation (they were spontaneously breathing) dropped about 25%. So the decrease in the work of breathing was substantial, with a really large decrease in P_{CO_2}. However these were animals with normal lungs—where it probably can be effective. That could be a potential use—not necessarily for patients on mechanical ventilation, but to avoid the need for intubation and ventilation. You can stick a catheter in and help the patient along, decrease their work of breathing and lower their P_{CO_2}. You may not have to intubate them and use mechanical ventilation.

1. Long SE, Menon AS, Kato H, Goldstein RS, Slutsky AS. Constant oxygen insufflation (COI) in a ventilatory failure model. Am Rev Respir Dis 1988;138(3):630-635.

Kacmarek: To follow up on Rich's (Branson) point—this is a technique that's not only difficult to do but also potentially dangerous. I do not think that we have sufficient data. Nobody should be using this unless they've had significant experience in the laboratory prior to going to the bedside and have the safeguards on the system that they're using to prevent any of the disastrous effects that might occur if an obstruction of the system occurred or if pressures were not properly monitored. When you use TGI you really have no idea of what's going on inside the lung because you can't measure the PEEP level, you can't measure plateau pressure (at least, not easily), and it's very difficult to determine what's happening. Just a caution—it's a technique that I consider promising, but I think we have neither the technical capabilities nor the monitoring capabilities to use TGI, except under very closely supervised, clinical protocols.

Hess: Exactly, as Alex (Adams) pointed out very well in his presentation, the ventilators were all designed to operate with no bias flow of any kind in the system. When you begin adding bias flow to the system, the ventilator has no way of handling it because the ventilator was not designed to operate in that environment.

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Dual-Control Modes of Mechanical Ventilation

Richard D Branson RRT and Neil R MacIntyre MD

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In Summary

Introduction

Since the late 1960s, mechanical ventilation of the adult patient has been accomplished primarily using volume-controlled ventilation (VCV). More recently, pressure-controlled ventilation (PCV) has been touted as an alternative to VCV. Each method has advantages and disadvantages. Recognition of the specific limitations of each technique has lead to the development of so-called dual-control modes in which breath delivery can be volume- or pressure-controlled, depending upon delivered tidal volume. In this paper, we discuss terminology, identify the limitations of VCV and PCV, describe dual-control modes, and review the literature concerning these modes.

Terminology

According to the system described by Chatburn, breath types and ventilator modes can be described according to control, phase, and conditional variables.[1-3] We present a brief review of this system to facilitate description of new modes.

Control Variables

The control variable is the variable (eg, pressure, volume, flow, or time) that the ventilator manipulates to accomplish inspiration. The control variable for a given breath can be identified by the fact that its behavior remains constant despite changes in ventilatory load. That is to say, the control variable remains constant regardless of pulmonary impedance.

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A version of this paper was presented by Mr Branson during the RESPIRATORY CARE Journal Conference, Mechanical Ventilation, Ventilatory Techniques, Pharmacology, and Patient Management Strategies, held in Cancún, Mexico, October 6-8, 1995.

The authors have no financial interest in the products described.

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All other pre-set variables are altered to allow the control variable to remain unchanged.

Phase Variables

According to Mushin et al,[4] the respiratory cycle has four phases: (1) change from expiration to inspiration; (2) inspiration; (3) change from inspiration to expiration; and (4) expiration. Phase variables, then, are the variables used by the ventilator to begin, sustain, and terminate each of the four phases. Phase variables are measured by the ventilator and include pressure, volume, flow, and time. For any given type of breath in a mode of ventilation, the trigger (change from expiration to inspiration), limit (inspiration), and cycle (change from inspiration to expiration) can be described.

Trigger—The trigger variable causes inspiration to begin. Ventilators can be flow- or pressure-triggered when the patient's inspiratory effort exceeds the set sensitivity or time-triggered according to the set ventilator rate. More simply, a breath can be triggered by the ventilator (time-triggered) or by the patient's inspiratory effort causing flow or pressure in the circuit to exceed the set sensitivity (patient-triggered).

Limit—The limit variable is the variable (pressure, volume, or flow) that the ventilator maintains at a pre-set value during inspiration, but which does not cause inspiration to end. For example, during pressure-support ventilation (PSV), inspiration continues even after the selected pressure limit has been reached and is terminated only after the flow decays.

Cycle—The cycle variable, when reached, terminates inspiration. Ventilator breaths may be time-cycled, volume-cycled, flow-cycled, or pressure-cycled. During PSV, inspiration is flow-cycled when inspiratory flow decays to a pre-set minimum flow or percentage of initial flow.

Conditional Variables

Conditional variables are those variables that, alone or in combination, are examined by the ventilator's control logic and used to determine what criterion will be used to manipulate a phase of the breath. A common conditional variable is patient effort. In the synchronized intermittent mandatory ventilation (SIMV) mode, the ventilator creates a timing window around the point at which a mandatory breath is due to be delivered (based on set frequency). If the ventilator senses a patient effort, the breath is delivered in synchrony with patient effort. If the patient does not initiate an inspiratory effort, the ventilator delivers the breath at the appropriate time to maintain the set frequency. In this example, the ventilator uses the conditional variable of patient effort to deliver either a patient-triggered or machine-triggered breath.

Using this information, any breath delivered by the ventilator or any ventilator mode can be described using the control, trigger, limit, cycle, and conditional variables.

Mandatory Breaths

When one describes breath types and modes, it is important to distinguish between mandatory and spontaneous breaths. The definitions are based on the trigger, limit, and cycle variables.

A mandatory breath is either triggered or cycled by the ventilator. Thus, a breath that is time-cycled and/or time-triggered is always a mandatory breath. A breath that is patient-triggered but remains machine-cycled is also a mandatory breath.

Spontaneous Breaths

A spontaneous breath is both triggered and cycled by the patient. Patient breaths during continuous positive airway pressure (CPAP) and PSV are spontaneous. In the case of PSV, all breaths are patient-triggered and patient-cycled, based on patient demand and pulmonary impedance.

Pressure Control vs Volume Control

Volume-controlled ventilation in either the continuous mandatory ventilation (CMV) or SIMV modes has been the ventilatory support method of choice in adults for the past 25 years. Early in the modern era of ventilatory support, ventilators were pressure-controlled (Bird Mark Series, Puritan Bennett PR series). The introduction of volume control and the guarantee of constant tidal volume (V_T) and minute volume ($\dot{V}_E$) delivery, made volume ventilators the ventilators of choice.

It is interesting to note that Fleming et al[5] found VCV to be superior to PCV in patients ventilated following major trauma. Fleming et al ventilated postoperative casualty patients in Vietnam using either a pressure-limited, time-cycled ventilator (Puritan Bennett PR-2, Bird Mark 7) or a volume-cycled ventilator (Emerson 3PV, Engstrom 300). They found that patients on VCV had a reduced incidence of atelectasis on chest radiograph, better oxygenation, and improved pulmonary compliance. Volume-cycled ventilators became the ventilators of choice.

In the last decade, increasing numbers of reports have suggested that PCV may be superior to VCV.[6-15] Because PCV is covered in a separate Journal Conference paper in the May issue of this Journal,[16] we present here only a brief comparison of PCV and VCV.

VCV has been used extensively in adults during the past 30 years, and, thus, a major advantage of VCV is caregiver experience. That is, the application of VCV and patient response have been well described. As mentioned previously, VCV allows a set V_T to be guaranteed, regardless of pulmonary impedance. Constant-volume delivery remains the most compelling factor in the selection of VCV. VCV also has the advantage of providing a linear increase in $\dot{V}_E$ as V_T is increased.

Disadvantages of VCV include excessive airway pressures that may lead to barotrauma, volutrauma, or hemodynamic compromise, and patient-ventilator dyssynchrony.[17] When the set inspiratory flow is less than patient demand, the consequent dyssynchrony may result in a work of breathing (WOB) equivalent to spontaneous breathing. This has been called flow starvation and is obviously undesirable. Studies by Marini et al,[18,19] Sassoon et al,[20] and Ward et al[21] have demonstrated that the single most important factor affecting patient WOB is the difference between set flow and patient flow demand. Figure 1 depicts a volume-cycled breath demonstrating the characteristic deformation of the pressure waveform seen during flow starvation. Next to this breath is depicted a manually-triggered breath unaffected by patient demand. We believe that comparison of these breaths is an ideal method for diagnosing flow starvation during VCV.

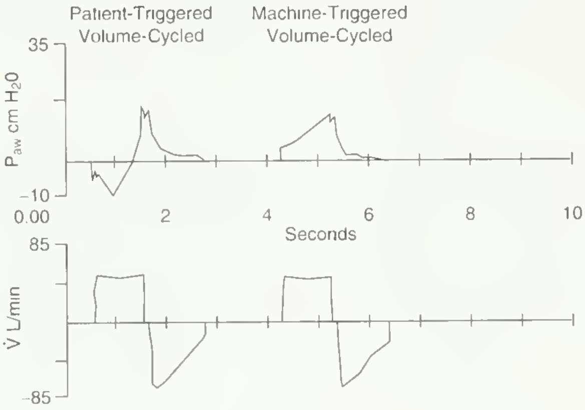


Fig. 1 Pressure waveforms demonstrating a machine-triggered, volume-cycled breath (right) and a patient-triggered, volume-cycled breath (left). The second breath demonstrates the characteristic deformation of the pressure waveform seen with patient-ventilator dyssynchrony.

Proponents point out that PCV (1) increases mean airway pressure by virtue of a rectangular inspiratory pressure pattern (descending-ramp flow waveform); (2) limits excessive airway pressure and, therefore, the risk of volutrauma and barotrauma; (3) improves gas distribution by exposing the lungs to the set peak inspiratory pressure (PIP) throughout the inspiratory time; and (4) reduces the WOB by providing a high initial flow that decreases as lung volume and pressure increase.[6-10] Studies in the last 5 years have concluded that PCV is superior to VCV (rectangular flow waveform),[6-10] equivalent to VCV (rectangular flow waveform),[11-14] and equivalent to VCV (descending-ramp flow waveform).[15]

Disadvantages of PCV include (1) variable V_T delivery as pulmonary impedance changes; (2) excessive volume delivery in the setting of improving pulmonary compliance, if volume alarms are not properly set; and (3) inconsistent changes in V_T with changes in PIP and positive end-expiratory pressure (PEEP). In the latter case, increasing PIP may fail to increase V_T, and in the presence of auto-PEEP V_T is diminished.[17-22] Table 1 compares VCV and PCV breath characteristics.

Table 1. Breath Characteristics for Volume-Controlled Ventilation and Pressure-Controlled Ventilation

Variable	Volume-Controlled Ventilation	Pressure-Controlled Ventilation
Trigger	Patient or machine	Patient or machine
Limit	Flow	Pressure
Cycle	Volume	Time or flow
Tidal Volume	Constant	Variable
Peak Pressure	Variable	Constant
Modes	Volume control-assist/control; volume-control-[synchronized] intermittent mandatory	Pressure control-assist/control; pressure control-[synchronized] intermittent mandatory; pressure-support

Combining Pressure & Volume Control

The concept of combining the positive attributes of pressure control (high flow changing to meet patient demand) and volume control ($\dot{V}_E$-guarantee) were first described by Amato et al in 1992.[23] A lung-model evaluation of a commercially available ventilator using a combined pressure-volume breath was described by MacIntyre and colleagues in 1994.[24] We (Haas et al[25]) evaluated a commercially available combined pressure-volume breath in 1995. These are the only evaluations currently available. We describe the theoretical concept, mechanical design, clinical experience, and individual ventilator manufacturer algorithms for combining pressure and volume breaths (ie, for accomplishing dual control).

The Pressure/Volume Breath

Amato et al's initial concept of volume-assured pressure-support ventilation, or VAPSV, has been adopted by both Bird Corporation and Bear Medical to provide dual control. Amato et al's[23] original description of VAPSV utilized two flow sources in parallel to achieve a high initial flow and a volume-constant breath. The first source provides flow with a rectangular pattern set by the clinician (10-120 L/min). This provides the constant volume. The second source of flow (up to 200 L/min) is set to reach a pre-set pressure limit. When the patient initiates a breath, both flow sources are activated. The second flow source rapidly delivers gas into the circuit until a pre-set pressure limit (set by the pressure support control) is reached. This flow then decreases to maintain constant pressure. If delivered V_T is greater than the pre-set minimum V_T then the breath is simply a pressure-supported breath. It is important to note at this point that although a minimum V_T is guaranteed, VAPSV allows V_T to exceed set V_T according to patient demand.

If V_T is less than the minimum set V_T, then as flow from the second source decreases, the primary constant source continues at the set flow until V_T is delivered. This causes the characteristic flow pattern demonstrating an initial decreasing flow waveform that is 'squared off' as the constant flow assures V_T delivery (Fig. 2).

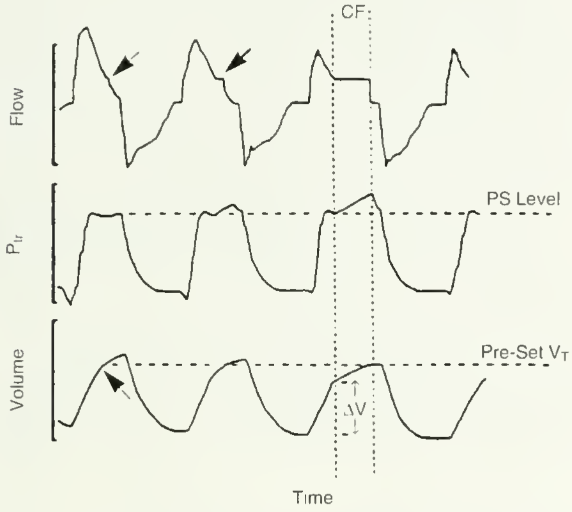


Fig. 2. Pressure and flow waveforms during volume-assisted pressure support in the study by Amato et al. CF = continuous flow. (Reprinted from Reference 23, with permission.)

Amato et al's description[23] intended VAPSV to be used as a substitute for conventional volume-controlled, assist/control ventilation. In this instance, the high initial flow aids in reducing the WOB and prevents flow dyssynchrony while the volume-guarantee assures constant $\dot{V}_E$. Dual control might be used as a 'safety net' during PSV. In this application, if patient effort is diminished during PSV, the ventilator flow decreases to a pre-set minimum and provides constant flow for the remaining portion of inspiration, until the desired V_T is delivered. The use of VAPSV in this manner has not been studied and although it assures V_T, it might have adverse consequences related to patient-ventilator synchrony. If during PSV, VAPSV is employed and diminishing patient effort causes flow to rapidly decelerate, then the sudden switch to constant flow might disrupt the patient's breathing pattern. This application of VAPSV deserves further study.

Clinical Experience with VAPSV

Amato et al's initial work[23] evaluated the effects of VAPSV on gas exchange and WOB in 8 patients in acute respiratory failure. They ventilated patients in a conventional volume-controlled mode with a rectangular flow waveform at a V_T of 10 mL/kg, inspiratory flow of 50 L/min, and an inspiratory pause of 0.15 seconds. During VAPSV, the minimum set flow was 25 L/min, and the pressure limit was set at 1-2 cm below the PIP during VCV. In this case, initial flow up to 200 L/min was delivered until the set pressure limit was reached, at which time flow began to decrease. If the minimum set V_T (10 mL/kg) had not been delivered when flow diminished to 25 L/min, then flow was held constant at that value until the target V_T was reached.

These investigators measured respiratory variables, blood gas values, and WOB indices during 40-minute periods of ventilatory support. The results from this study are shown in Table 2. Amato et al[23] found that VAPSV reduced the WOB, expressed as joules/L, by almost 50% [mean (SD) = 0.53 (0.48) vs 0.99 (1.23) J/L] compared to VCV, reduced airway resistance, and increased dynamic compliance. The authors attributed this to the higher inspiratory flow with VAPSV [60 (40) vs 46 (8) L/min], which resulted in a larger V_T, shorter inspiratory time, and reduced auto-PEEP.

Table 2. Respiratory Variables Measured during Volume-Assisted Ventilation (VAV) and Volume-Assisted Pressure-Support Ventilation (VAPSV), from Amato et al[23]

Variable	VAV*	VAPSV	p Value
V_T, L	0.59 (0.16)	0.72 (0.23)	< 0.02
f, breaths/min	23.5 (7.1)	21.1 (0.23)	NS
$\dot{V}_E$, L	13.6 (4.1)	14.2 (5.1)	NS
t_I, s	0.79 (0.12)	0.76 (0.15)	NS
V_T/t_I, L/min	46.0 (8.4)	59.6 (10.2)	< 0.01
P_{tr}, cm H_2O	34.9 (11.7)	33.0 (11.6)	NS
$C_{dyn\text{-}rs}$, mL/cm H_2O	23.8 (9.2)	31.0 (16.1)	< 0.05
t_{rs}, cm $H_2O \cdot s \cdot L^{-1}$	46.7 (15.3)	35.3 (19.7)	< 0.01
WOB/L, J/L	0.99 (1.23)	0.53 (0.48)	< 0.03
WOB/min, J/min	12.9 (10.5)	7.8 (6.5)	< 0.05
PTPb, cm $H_2O \cdot s$	6.91 (3.63)	3.31 (2.83)	< 0.05
$P_{0.1\text{-}tr}$, cm H_2O	4.9 (3.6)	2.8 (2.1)	< 0.05
$P_{0.1\text{-}es}$, cm H_2O	5.0 (4.2)	2.5 (2.0)	< 0.10
P_{aO_2}, torr	187 (63)	214 (57)	NS
P_{aCO_2}, torr	41.4 (6.7)	38.9 (8.1)	NS

*Values are mean (SD). V_T = tidal volume; f = breathing frequency; T_I = inspiratory time; P_{tr} = tracheal pressure; $C_{dyn\text{-}rs}$ = dynamic compliance of the respiratory system; l_{rs} = inspiratory resistance; WOB = work of breathing; PTPb = pressure time product/breath; $P_{0.1\text{-}tr}$ = occlusion pressure at 100 ms measured in the trachea; $P_{0.1\text{-}es}$ = occlusion pressure measured at 100 ms in esophagus; P_{aO_2} = partial pressure of oxygen in arterial blood. P_{aCO_2} = partial pressure of carbon dioxide in arterial blood

Amato et al[23] suggested that VAPSV improves patient-ventilator synchrony by matching ventilator flow output to patient demand and reduces airway pressures by delivery of high flow during early inspiration and lower flows at end expiration. We can criticize Amato et al's work because of the relatively low peak flow during VCV (46 L/min) and the significantly larger V_T delivered during VAPSV (0.72 L vs 0.59 L), a 23% increase. It can be argued that if V_T and inspiratory flow during VCV are equivalent to that during VAPS, no differences would be seen. However, if both V_T and flow are increased during VCV, PIP rises dramatically. In addition, regardless of the chosen peak flow during VCV, fluctuating patient demands appear to be better met by VAPSV than by VCV.

In a recent paper,[25] we evaluated a commercially available version of VAPSV provided by the Bird 8400STi. Our goal was to verify the findings of Amato et al, while attempting to control V_T and inspiratory flow during VCV. This was accomplished by setting the pressure limit during VAPSV to provide a V_T equivalent to VCV and by attempting to match set inspiratory flow to patient demand during VCV. We studied 17 patients during 40-minute periods of VCV, VAPSV, and a second period of VCV. During each period, respiratory variables including V_T, frequency, minute ventilation, PIP, auto-PEEP, WOB, pressure-time product (PTP), and transpulmonary pressure generated during the first 100 milliseconds of inspiration ($P_{0.1}$) were continuously monitored. Arterial blood was analyzed for blood gas values and pH at the end of each 40-minute period.

Results from our study[25] are shown in Tables 3 and 4. We found a significant reduction in PTP with VAPSV compared to VCV (47 vs 73 cm $H_2O \cdot s \cdot min^{-1}$) and statistically significant reductions in $P_{0.1}$ and esophageal pressure. A trend toward lower auto-PEEP and WOB during VAPSV did not become clinically important. Despite our attempts to maintain constant V_T over the course of the 40-minute study period, V_T drifted upward by 5%. This is a statistically significant increase but may have little clinical impact. However, we think this is an important observation, in that as patient demands vary, the ability to increase V_T may play an important role in controlling the sensation of breathlessness.[26]

Our study[25] is also open to criticism. We attempted to study patients during uninterrupted periods. As a consequence, the changes in patient demand typically seen as a result of pain, anxiety, and patient-care procedures were excluded from our analysis. We believe that during times of stress, VAPSV may prove to be a more valuable technique than is suggested by the results of our study of resting patients.

We, like Amato et al,[23] have learned that the initial set pressure limit should be less than PIP and is best approximated by using as the pressure-support level the plateau pressure seen during VCV. We also found that the minimum flow setting can affect patient synchrony. We set the minimum flow at 40 L/min as compared to Amato et al's 25 L/min. We believe it is important to have the constant flow activate earlier in the inspiratory cycle. If the flow is set too low, the possibility of

Table 3. Respiratory Mechanics Measurements from Study of Volume-Assured Pressure-Supported Ventilation by Haas et al[25]

Variable	$\dot{V}_{set-1}$*	$\dot{V}_{pd}$	$\dot{V}_{set-2}$	p Value
P_{es}, cm H_2O	12.2 (3.6)	10.7 (4.1)	13.9 (5.4)	a, c[†]
PTP cm $H_2O \cdot s \cdot min^{-1}$	73 (59)	47 (49)	92 (78)	a, c
C_{dyn}, mL/cm H_2O	64.7 (54.8)	68.4 (65.5)	68.4 (63.4)	0.168
R_{aw}, cm $H_2O \cdot s \cdot L^{-1}$	11.0 (6.5)	12.3 (9.1)	11.7 (8.0)	0.175
$WOB_{patient}$, J/L	0.52 (0.34)	0.43 (0.70)	0.58 (0.60)	0.079
$WOB_{ventilator}$ J/L	2.01 (0.81)	2.16 (0.80)	2.10 (0.81)	0.288
t_I/t_{tot}	0.30 (0.07)	0.28 (0.07)	0.30 (0.07)	a
$P_{0.1}$, cm H_2O	2.27 (1.02)	1.94 (1.31)	2.65 (1.36)	a

*Values are mean (SD); $\dot{V}_{set-1}$ = ventilator set flow in Baseline 1; $\dot{V}_{pd}$ = patient-determined flow; $\dot{V}_{set-2}$ = ventilator set flow in Baseline 2. P_{es} = esophageal pressure; PTP = pressure time product; C_{dyn} = dynamic compliance; R_{aw} = airway resistance; WOB = work of breathing; t_I = inspiratory time; t_{tot} = total length of respiratory cycle; $P_{0.1}$ = occlusion pressure at 100 ms.

Table 4. Mean (SD) Values for Ventilatory Variables from Study of Volume-Assured Pressure-Supported Ventilation by Haas et al[25]

Variable	$\dot{V}_{set-1}$*	$\dot{V}_{pd}$	$\dot{V}_{set-2}$	p Value
$\dot{V}_{max}$, L/min	54.2 (9.6)	62.9 (10.1)	54.9 (10.2)	a, b, c[†]
$\dot{V}_E$, L/min	11.8 (3.1)	11.5 (3.2)	12.7 (3.6)	a, c
V_T, mL	816.0 (22.0)	857.0 (23.0)	812.0 (22.0)	a
f, breath/min	15.2 (5.6)	14.5 (6.7)	16.5 (6.4)	a, c
$PEEP_I$, cm H_2O	2.9 (1.8)	2.5 (2.2)	3.3 (2.8)	0.104
PIP, cm H_2O	40.2 (10.5)	40.6 (11.9)	40.2 (11.9)	0.084
P_{aw}, cm H_2O	14.9 (5.5)	15.6 (6.0)	15.2 (6.0)	0.256

*Values are mean (SD); $\dot{V}_{set-1}$ = ventilator set flow in Baseline 1; $\dot{V}_{pd}$ = patient-determined flow, $\dot{V}_{set-2}$ = ventilator set flow in Baseline 2. $\dot{V}_{max}$ = maximum flow; $\dot{V}_E$ = expiratory flow; V_T = tidal volume; f = breathing frequency; $PEEP_I$ = intrinsic, or auto-PEEP, PIP = peak inspiratory pressure; P_{aw} = airway pressure.

a = $p < 0.05$ using repeated-measures analysis of variance (ANOVA); b,c = $p < 0.0167$ for post-hoc analysis, b comparing $\dot{V}_{pd}$ to $\dot{V}_{set-1}$, and c comparing $\dot{V}_{pd}$ to $\dot{V}_{set-2}$.

prolonged inspiratory time and air trapping is increased. A typical set of flow and pressure curves for VCV and VAPS are shown in Figure 3. Table 5 describes VAPSV operation with varying V_T.

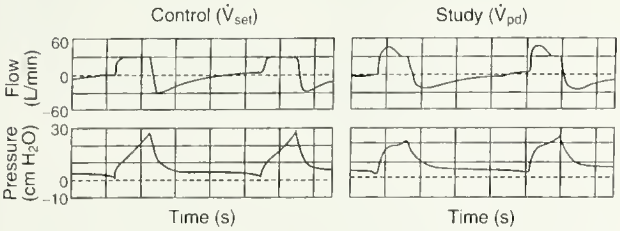


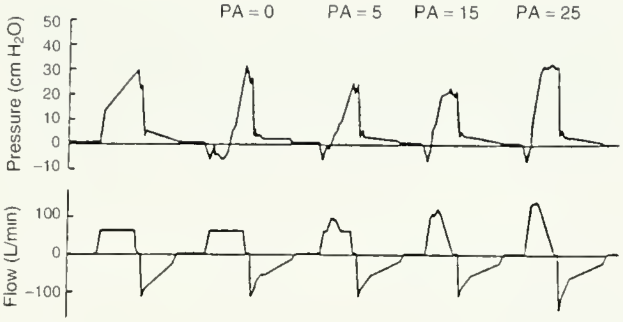
Fig. 3. Flow and pressure waveforms during volume-controlled ventilation and volume-assisted pressure support in the study by Haas et al. (Reprinted from Reference 25, with permission.)

Table 5. Characteristics of Volume-Assisted Pressure-Support Breaths Based on Relationship between Delivered and Set Minimum Tidal Volume from Study by Haas et al[25]

Variable	Condition Delivered V_T > Set V_T	 Delivered V_T < Set V_T
Control	Pressure	Pressure → Volume
Trigger	Patient (pressure or flow)	Patient (pressure or flow)
Limit	Pressure	Pressure → Flow
Cycle	Flow	Volume

MacIntyre and others[24] evaluated the VAPSV function of the Bear 1000 (so-called Pressure Augmentation) in a lung model at varying degrees of simulated patient demand. They found that the VAPSV-type breath allows for more effective matching of ventilator output to simulated patient demand than traditional VCV, regardless of flow waveform. Figure 4 depicts five simulated breaths during Pressure Augmentation. The first breath is a mandatory breath delivered to the passive lung model; the second breath is a breath delivered at the same V_T during a simulated patient demand of 90 L/min. The next three breaths show the addition of 5, 15, and 25 cm H_2O of Pressure Augmentation. As pressure is increased, the waveform takes on a more normal appearance (similar to the passive breath) suggesting improved matching of ventilator flow delivery to patient demand. MacIntyre et al[24] suggested that this breath type could replace both volume- and pressure-controlled breaths on future ventilators. Clinicians could then select the VAPSV breath with a high pressure-limit to provide enhanced patient-ventilator synchrony or at a lower pressure limit to assure constant V_T and inspiratory time.

Although few clinical trials using VAPSV have been reported, this breath delivery technique appears to reduce the WOB and improve synchrony while being easy to use and relatively free of complications.



Patient.					
• Flow demand (L/min)	0	120	120	120	120
• Volume delivered (mL)	500	500	500	500	500
• Work/breath (J)	0	0.378	0.373	0.343	0.280

Fig. 4. Pressure waveforms during volume-controlled ventilation and the application of Pressure Augmentation (PA). See text for details. (Reprinted from Reference 24, with permission.)

Commercial Systems for Dual Control

Both the Bird 8400STi and Bear 1000 allow dual-control of breaths. The 8400STi identifies this feature as VAPS and the Bear 1000 identifies it as Pressure Augmentation.

Bird 8400STi: The VAPS modification of the 8400STi allows dual breath control during SIMV, Assist/Control, and PSV. VAPS is activated by depressing the membrane pad labeled VAPS, and the clinician is alerted to VAPS operation by a light above the membrane pad. During VAPS operation, the V_T control sets the minimum delivered V_T, and the peak flow represents the flow to be delivered if the V_T is less than the minimum value. The pressure support control is used to set the pressure limit. As the pressure support level is increased, initial inspiratory flow becomes higher and V_T larger.

Figure 5 depicts the three possible types of breaths delivered during VAPSV. The first breath (A) represents a typical, synchronous, pressure-limited, time-cycled breath. This type of breath allows for improved synchrony but cannot guarantee a constant V_T. The second breath (B) shows a VAPSV breath when patient demand is reduced. The pressure support level is 25 cm H_2O, the peak flow is set at 40 L/min, and the desired V_T is 800 mL. The start of the breath shows a rapid increase in flow followed by an initial decrease. When flow decreases to the peak flow setting and delivered V_T is less than set V_T, the breath transitions to a volume-controlled breath, and flow remains constant until the V_T is delivered. Note that as flow remains constant, peak pressure exceeds the pressure support level. In Breath C, lung compliance is reduced, and the breath transitions to a volume-controlled breath earlier and requires a longer inspiratory time to reach the desired V_T. In this situation, PIP is higher and the ratio of inspiratory-to-expiratory time is affected. Caution should be exercised to assure that sufficient expiratory time is provided. In Breath D, increased patient demand allows a V_T greater than set, and the breath is a pressure-limited, flow-cycled breath (pressure support). The pressure support level is adjustable from 1 to 50 cm H_2O, and the peak flow setting from 10 to 120 L/min.

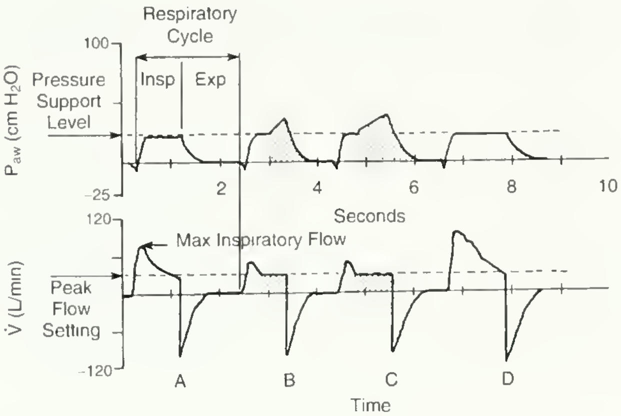


Fig. 5. Waveforms demonstrating: A. a pressure-limited, time-cycled breath; B. a volume-assisted pressure-supported (VAPS) breath, demonstrating transition from pressure to volume control; C. a VAPS breath demonstrating transition from pressure to volume control due to reduced lung compliance; and D. a pressure-supported breath delivered during VAPS when delivered V_T is > set V_T.

Bear 1000: The Bear 1000 allows dual control by activating the Pressure Augmentation function. Pressure Augmentation is active in the Assist/Control, SIMV, and PSV modes. Pressure Augmentation is activated by pressing the PRES AUGMENT control, setting the PRES SUP/INSP PRES control to the desired level, and setting the minimum V_T. The design of the Pressure Augmentation algorithm is similar to the VAPSV algorithm of the 8400STi. A flow diagram demonstrating the control logic of the Pressure Augmentation function of the Bear 1000 is shown in Figure 6.[27]

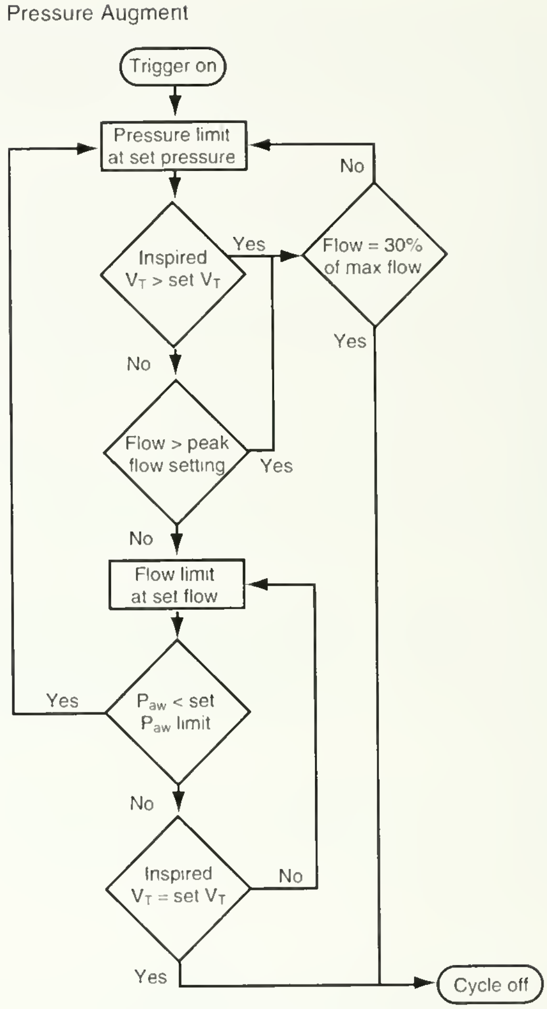


Fig. 6. Flow diagram for Pressure Augmentation function of the Bear 1000 ventilator. (Reprinted from Reference 27, with permission.)

Volume Support & Pressure-Regulated Volume Control

Another approach to combining pressure and volume breaths has been accomplished by the Siemens 300 ventilator. These two techniques are not new modes or new breath-delivery types but simply closed-loop control of two pressure-limited modes—pressure support and pressure-limited, time-cycled ventilation.

Volume Support

Volume support (VS) is pressure-supported ventilation that uses V_T as a feedback control for continuously adjusting the pressure-support level. VS is selected with the mode selector switch and the desired V_T is set. The ventilator initiates VS by delivering a "test breath" with a PIP of 5 cm H_2O when a patient effort is sensed. The delivered V_T is measured, and total system compliance is calculated. The three breaths following are delivered at a PIP of 75% of the pressure calculated to deliver the minimum V_T. Each of the breaths following uses the previous calculation of system compliance to manipulate PIP to achieve the desired V_T. From breath to breath, the maximum pressure change is 3 cm H_2O and can range from 0 cm H_2O above PEEP to 5 cm H_2O below the high-pressure alarm setting. Because all breaths are pressure-supported breaths, cycling normally occurs at 5% of the initial peak flow. A secondary cycling mechanism is activated if inspiratory time exceeds 80% of the set total cycle time. A schematic of volume-supported operation is shown in Figure 7. A diagram demonstrating the control logic of volume support is shown in Figure 8.

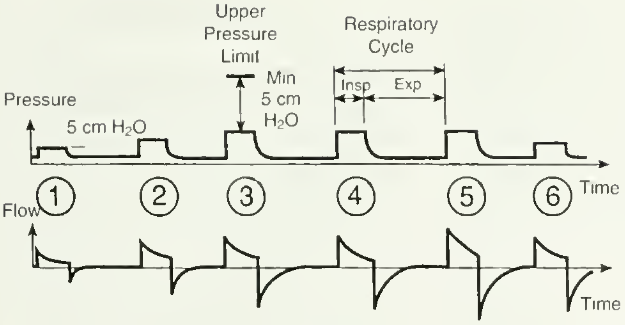


Fig. 7. The volume-support mode: first breath is a test breath with a maximum pressure level of 5 cm H_2O. The pressure changes breath by breath until pre-set tidal volume (V_T) is obtained. Maximum available pressure level is 5 cm H_2O below pre-set upper pressure limit. Breaths are delivered with the pre-set minimum volume at the pre-set frequency and during pre-set inspiration time. When measured V_T corresponds to pre-set value, pressure level remains constant. If measured volumes increase above pre-set minute volume, pressure level decreases in small increments until pre-set volumes are delivered. The ventilator is continuously adapting the inspiratory pressure to changes in the volume-pressure relationship. (Diagram courtesy of Siemens).

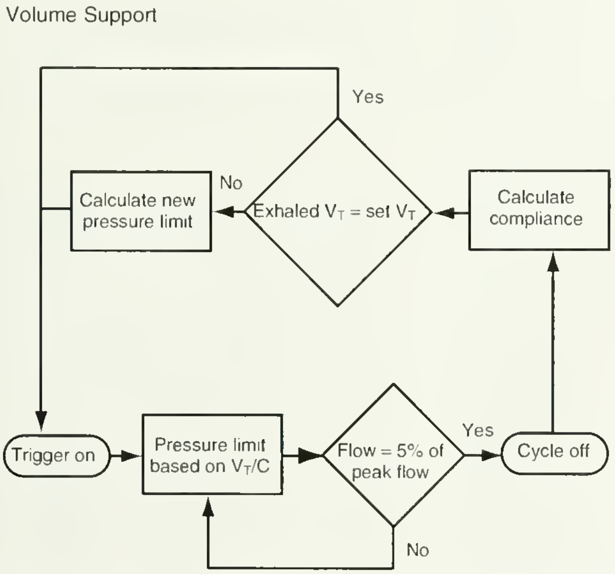


Fig. 8. Control logic of the volume-support mode of the Siemens 300 ventilator. (Reprinted from Reference 27, with permission.)

Pressure-Regulated Volume Control (PRVC)

PRVC, available on the Siemens 300, is pressure-limited, time-cycled ventilation that uses V_T as a feedback control for continuously adjusting the pressure limit. PRVC is selected on the mode selector switch, and the desired V_T is set. As with volume support, a test breath is delivered, and total system compliance is calculated. The next three breaths are delivered at a pressure limited to 75% of that necessary to achieve the desired V_T based on the compliance calculation. The ensuing breaths increase or decrease the PIP at < 3 cm H_2O per breath in an attempt to deliver the desired V_T. The PIP fluctuates between 0 cm H_2O above the PEEP level to 5 cm H_2O below the upper pressure-alarm setting. Figure 9 is a flow diagram for PRVC control logic and Figure 10 depicts the pressure and flow waveforms during PRVC.

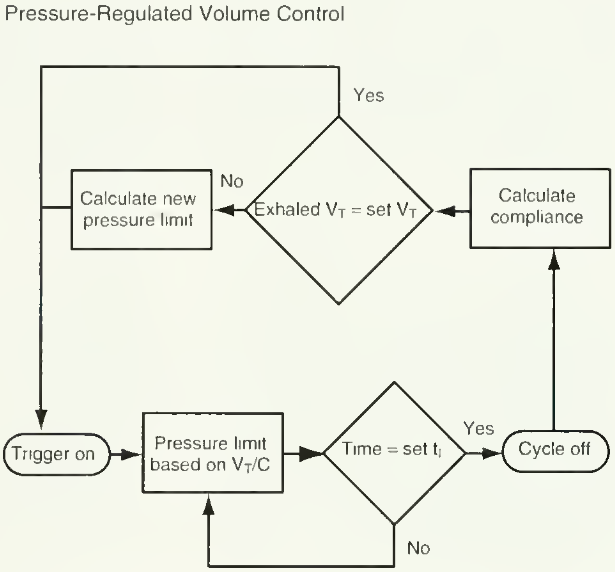

Pressure-Regulated Volume Control
Yes
Calculate new pressure limit
No
Exhaled V_T = set V_T
Calculate compliance
Trigger on
Pressure limit based on V_T/C
Time = set t_i
Yes
Cycle off
No

Fig. 9. Control logic of the pressure-regulated volume control mode of the Siemens 300. (Reprinted from Reference 27, with permission.)

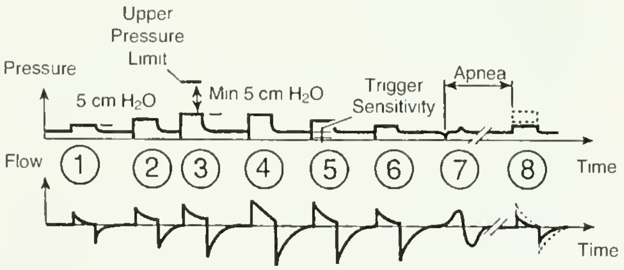


Fig. 10. The PRVC mode: patient effort initiates a test breath with a maximum pressure level of 5 cm H_2O. If the support is not enough to give adequate tidal volumes (V_Ts), pressure support is added stepwise. Maximum available pressure support is 5 cm H_2O below pre-set upper pressure limit. If pressure support generates volumes greater than the pre-set V_T, pressure support is lowered stepwise until satisfactory volumes are achieved. Every breath is initiated by patient effort. If the patient has the capacity to breathe adequately with no support, the ventilator permits true spontaneous breathing with monitoring of achieved volumes. If the apnea-alarm time limit passes with no new breath triggered, the apnea alarm is activated and the ventilator automatically switches to mandatory pressure-regulated volume-controlled ventilation (PRVC). The ventilator continues PRVC until the apnea alarm is manually reset; then it returns to volume-supported ventilation.

Clinical Experience

To date (1996), no published studies report on the use of either VS or PRVC. Both these modes are similar to mandatory minute ventilation (MMV) except that V_T, as opposed to $\dot{V}_E$, is used as the feedback signal. It may be that VS allows patients on PSV to have a constant V_T and, thus, possibly to wean themselves. As patient strength and effort improve, the ventilator automatically reduces support. On the other hand, patients might reduce their efforts and the ventilator support might increase to a point where muscle atrophy develops and weaning is delayed. Current therapy of PSV typically relies on changes in respiratory rate and V_T. If V_T were to remain constant, evaluation of patient response would require modification. Both VS and PRVC require clinical evaluation.

In Summary

Ventilator manufacturers have attempted to answer the concerns of clinicians related to PCV and VCV by providing dual control of breaths or by using closed-loop control. The current literature suggests that VAPSV is a safe and effective method to improve patient-ventilator synchrony and reduce the WOB. However, results have been reported in only 17 patients ventilated with a commercial device.[25,26] The clinical utility of PRVC and VS remains to be elucidated.

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Branson Discussion

Kacmarek: When *wouldn't* you want to use pressure-limited, volume-cycled ventilation (VAPSV)? It seems to me that this just makes what we have better, and it's difficult, as you well know, to set flows and volumes to meet patient demand 24 hours a day. I see no reason why—if you're going to choose a volume target—that you would not use VAPSV. I mean, it's available. I think it should be used in every single patient you ventilate with a volume target.

Branson: I agree, Bob, but then I'll give you a couple of reasons why you might not. When Carl (Haas) and I were doing this study,[1] we specifically tried to make sure that before we made any measurements, we set the flow appropriately. We tried to make sure that the patients had no auto-PEEP and no flow dyssynchrony, and that we had what we thought was pretty reasonable matching of the ventilator to patient demand. But I think you're absolutely right. The therapist can't sit there and make that change when the patient's getting a bath, when the patient's getting an x-ray, when the patient's sedation has worn off, or when the patient's sedation is excessive. So, I think there are only two reasons not to use VAPSV—first, you can lull yourself to sleep by having it on all the time and end up with a patient always getting a bigger tidal volume than you wanted on pressure support, and you may actually get to a point where you've unloaded their muscles for too long. Second, in cases of radically reduced impedance because of pulmonary edema or because of bronchospasm, you could have long inspiratory times that could lead to air trapping. But, I think other alarms would be acting up if that were the case. I think I probably agree with you that if it's available and it's just a button that you push on or off, there is no really good reason not to use it.

Kacmarek: I think the limitations that you've just described are important. There are problems that can occur with any patient we ventilate if you don't pay attention to what's going on. Why should we even have standard volume-cycled breaths on the ventilators—now that we have this approach available?

MacIntyre: I'm going to mostly agree, but maybe clarify just a little bit. When you talk about the combined pressure-limited, volume-cycled mode, people usually think of it as being used in two situations. The first is for the patient to whom you want to give total mechanical support to, whom we traditionally give volume assist/control. I think, under these circumstances, Bob, that I'm in 100% agreement with you. You should think of this approach as actually just providing rapid variable flow instead of the usual fixed flow, and if you believe in improved alveolar gas mixing with this flow pattern, that's fine. It certainly is going to be more synchronous. So, under those circumstances—applying it during total support where we traditionally use assist/control—it just makes a lot of sense. Our approach under these circumstances is usually to set the pressure limit somewhere near the plateau pressure. I'm less clear on the other situation in which people talk about pressure-limited, volume-cycled breaths being used as a 'safety net' for pressure-support weaning. The idea here is to have the patient on partial support when you're trying to wean with pressure support. The usual approach, of course, is just to drop the pressure level. However, with this breath, the volume-cycled feature functions as the safety net so that the patient's tidal volume can't fall out from underneath him. On the surface, that sounds good. But if you think about it, I'm not so sure it is good because what it means is that as you're trying to wean, you have this safety net that won't let tidal volumes go below a certain arbitrary level, and you may never know whether they're progressing or not progressing. They'll always be, if you will, caught by this backup tidal volume. So, I'm less clear and less comfortable using it as a safety net for pressure-support weaning. I am very comfortable using it as a variable-flow approach to total support.

Kacmarek: I didn't mean to imply that this should be the mode of choice in *all* patients we ventilate, but, if you choose a volume-targeted approach to ventilation, for whatever reason, I see no reason to choose that without VAPSV, if it is available.

MacIntyre: Respiratory therapists sometimes ask How on earth are we going to get physicians to go along with this, and how do we get the order written for this? My approach is: don't tell them and don't ask for an order. The reason I say that, Bob, is because I think of it again as a flow adjustment. Respiratory therapists ordinarily do not ask for flow orders. It's usually left to the discretion of a good respiratory therapist to set the flow pattern during volume-targeted assist/control, and if you think of this pressure-limited feature on the front end of the breath as just being an extension of that flow setting (ie, going to a rapid decelerating flow instead of the traditional fixed flow) I see no reason to force an order in the order book for that.

Kacmarek: Well, of course, Neil, I agree with your comment. We had the same experience when we started to switch from pressure-triggering to flow-triggering. The most difficult thing was the use of the term flow-by, which implied a mode, and it was very difficult to ensure that staff, particularly residents, understood what you were talking about.

Branson: I hated to call VAPSV a mode—although that's really what people are calling it—because you can use it in IMV. You put the patient in IMV, and you just select the VAPS button—on or off. Then you adjust the pressure-support, and you're still delivering IMV

1. Haas CF, Branson RD, Folk LM, Campbell RS, Wise CR, Davis K Jr, et al. Patient-determined inspiratory flow during assisted mechanical ventilation. Respir Care 1995;40(7):716-721.

at the tidal volume that's been set on the ventilator. So, I agree. I'm not so sure that it's not just like flow-triggering, Bob. I mean I'm not so sure that VAPSV requires an order. We probably ought to just invoke it in every single circumstance in which we're trying to get constant-volume ventilation.

Peruzzi: My comment relates to what Neil alluded to earlier. When we're thinking of using pressure-support ventilation—and we use it a great deal in weaning patients who have compliance problems (recovering ARDS or the like)—we tend to pay more attention to respiratory rate as an indicator of work of breathing and give only cursory attention to tidal volumes. We use the patient's spontaneous rate to tell us how to titrate the level of pressure support. So, while I agree with you to a large extent, that VAPSV is a flow maneuver and doesn't require an order to implement, I do think that it requires appropriate communication and discussion with the clinicians, so that everyone is on the same wavelength and is clear as to where we do and do not want to take a volume-directed approach, and make certain that people are doing things appropriately.

MacIntyre: Bill, I think we are talking about two different things. What I was referring to and what Bob's referring to is when you have a patient for whom you want volume-targeted ventilation, it makes sense to add the combined pressure-limited, volume-cycled function. Moreover, I don't see a reason to require an order for it. What you're describing during the weaning process, however, is not a pure, volume-targeted approach, and I agree with you completely. Respiratory rate is critical and, in fact, if you have this volume safety net underneath patients, then tidal volumes may never fall low enough to tell you that they're not ready to be weaned further.

Branson: I think the other thing that we have noticed is that if you invoke VAPSV in the pressure-support mode and you set the peak flow at 50 L/min, if flow rapidly decelerates to the fixed flow, it is the patient's way of telling you that he's getting ready to exhale. When, all of a sudden, the ventilator stops and just keeps the flow constant, even though the patient may be wanting to exhale, you actually see some patients become wide-eyed as if to say, "Oh my God, what happened to the ventilator—I'm not in control any more." So, I'm much more comfortable with VAPSV in the volume-control mode than I am as a backup to the pressure-support mode.

Haas: When you notice that the preset VAPS flow regularly turns on, do you make adjustments to minimize this occurring—such as increasing the pressure-support level or decreasing the set flow rate?

Branson: Well, in volume-control, if you notice that the flow keeps squaring off to make sure the tidal volume is delivered, should you just increase the pressure-support level to make all the breaths more like pressure-support breaths?

Haas: Yes, and should you also consider decreasing the flowrate at which VAPS cycles on, so that it's more like a pressure-support breath?

Branson: We talked about some of this during the study.[1] If you see it happening all the time— on every single breath, the continuous flow is being invoked—that suggests to me that maybe you ought to increase the VAPSV or pressure-augmentation level to further reduce the work of breathing. If you decrease the set minimum flow limit, I'm not sure what the effect would be.

1. Haas CF, Branson RD, Folk LM, Campbell RS, Wise CR, Davis K Jr, et al. Patient-determined inspiratory flow during assisted mechanical ventilation. Respir Care 1995;40(7):716-721.

MacIntyre: I recommend raising the pressure limit, rather than lengthening the inspiration. I think the problem you run into, when people do set the inspiratory flow very, very low, is an inspiratory time that can be quite long. You shouldn't run into too much problem with air trapping if your inspiratory time and flow settings are in more conventional ranges.

East: Maybe I'm naive, but explain the advantages and disadvantages of the volume-support approach that Siemens took as opposed to the VAPSV approach that Bird took. What are the pros and cons? You seem to believe that VAPS is better than volume-support.

Branson: Maybe I do. Maybe that's because I'm used to it, and I've never used volume-support. I think that VAPS and Pressure Augmentation are really changing breath delivery; whereas, volume-support and pressure-regulated volume control are just simply increasing or decreasing the pressure limit.

East: Yes, but they also change the peak flows.

Branson: I'm not sure that is true, Tom (East)—that the peak flow always changes just because you increase the pressure. I don't know that one is better than the other, and the problem is there's no literature on volume-support or pressure-regulated volume-control. It's just simply, pressure-regulated volume-control and volume-support. It's analogous to having me stand there and watch the tidal volume signal and turn it up or turn it down, which is, I think, a little different from the breath-delivery techniques that we're talking about. If all of a sudden the patient's demand for flow and pressure-regulated volume control were to double, and I can only increase the pressure in 3 cm H_2O increments, then by the time I meet the patient's demand, demand may be back down again. That may be a little bit of the problem.

East: But, that can also be an advantage. You may not want to respond to those transient demands.

Kacmarek: But, I think you do. You have a dyssynchronous patient-ventilator system if you don't. What you truly have with VAPS is a volume-targeted breath where your primary interest is maintaining consistent tidal volume delivery; whereas, with volume-support, your primary interest—the variable that's targeted—is the pressure-support level. Tidal volume is a secondary variable with volume-support. You adjust the pressure-support level as tidal volume varies, but you are not guaranteed a constant tidal volume. With VAPS, you are guaranteed a constant tidal volume. So, it really depends on your focus. These manufacturers just came at it from two different approaches—one focusing primarily on pressure and the other one focusing primarily on volume.

East: But VAPS doesn't guarantee a *constant* value. It guarantees a *minimum* volume, right?

Kacmarek: Exactly. But you can exceed it.

East: Yes, you can exceed it. So that's another difference, pro or con, I guess.

Kacmarek: Again, it looks at the volume. The primary target of VAPS is volume, whereas with volume-support, the primary target is pressure.

East: Well, it's also volume. The difference with volume-support is that if you are above the desired volume, it would turn down the pressure-support level.

Kacmarek: That's true.

East: I don't know whether it's good or bad, but it's a difference.

Kacmarek: Neither do I. There are no data.

Branson: I don't know either. I think my sentiment is that probably one really is truly a mode or a breath-delivery technique that can be either strictly pressure or strictly volume, depending on the patient's needs at that breath moment. The other one is not really a new breath-delivery technique. It's just pressure-support and it's just pressure-control with a closed-loop algorithm based on tidal volume.

East: I think a lot of it is semantics. You could argue that VAPSV is nothing but pressure support augmented by constant-flow if volume is low. It's not a big deal, from a technologic point of view.

Branson: I think it's technically different from any other breath that we have. It changes on a breath-to-breath basis. One breath can be a totally pressure-supported breath, one breath can be part pressure-support with a volume cycle. Again, this is just in terms of describing new breath-delivery techniques. That *is* a new breath-delivery technique, whereas pressure support is pressure support. If you 'close-loop' it, that's new, and it may be advantageous, but it's not a new breath-delivery technique. I guess that was my point.

MacIntyre: Rich, I think the important point is that there are more similarities than differences. The reason I say that is because both techniques are trying to utilize features of a pressure-limited breath and, at the same time, give you some type of volume guarantee. Whether you do it the Servo (Siemens) way, which is to adjust it gradually over several breaths, or whether you use it the Bird and Bear way, which is to do it on that particular breath, I'm not sure how much difference there really is. Again, I think the message from this particular discussion is that there are more similarities than there are differences, at least in conceptual approaches to this problem, among these 3 ventilators.

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Extracorporeal and Intracorporeal Techniques for the Treatment of Severe Respiratory Failure

Barbara G Wilson MEd RRT

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Introduction

Despite the continuous growth of our knowledge and skill in treating acute respiratory distress syndrome (ARDS) in adults, the mortality rate has remained at greater than 60% through the past 15 to 20 years.[1-6] Management strategies for mechanical ventilation have played a central role in the support of these challenging patients, but the conventional practice of ventilating with large tidal volumes necessitating high inflation pressures to maintain near-normal arterial blood gases has been shown to cause severe lung injury.[7-15] Newer, pressure-limited ventilatory strategies[16] appear promising but have yet to demonstrate widespread changes in complication rates and patient survival. The failure to consistently prevent complications and improve survival has stimulated other investigators to explore alternative forms of support to improve oxygenation and ventilation.[17-22]

Technologic advances of the 1970s allowed introduction of several extracorporeal and intracorporeal techniques for improving oxygen delivery in an attempt to minimize the deleterious effects of mechanical ventilation. The term extracorporeal support describes devices for gas exchange outside the body and includes two basic techniques: extracorporeal membrane oxygenation (ECMO) and low-frequency positive-pressure ventilation with extracorporeal carbon dioxide removal (LFPPV-ECCO$_2$R). Intracorporeal support requires the surgical implantation of mechanical devices within the body for augmentation of blood gas transfer in patients with reversible respiratory failure. Several types of intracorporeal devices have been proposed: first and most well known is the intravascular oxygenator (IVOX), which is placed inside the lumen of a large blood vessel (vena cava); others involve in-situ replacement of natural lung using right-ventricle-driven lung-bypass devices or arteriovenous shunt exchangers.[23] The IVOX is the most thoroughly researched intracorporeal device,

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A version of this paper was presented by Ms Wilson during the RESPIRATORY CARE Journal Conference, Mechanical Ventilation: Ventilatory Techniques, Pharmacology, and Patient Management Strategies, held in Cancún, Mexico, October 6-8, 1995.

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and laboratory and human clinical trials have been completed with it. The other intracorporeal devices remain experimental and limited to bench or animal investigation. In this paper, I review the clinical practice and patient outcomes of intracorporeal and extracorporeal support for adults with ARDS and provide readers with information that should help them determine whether such methods of oxygenation support should be considered for their patients.

Extracorporeal Membrane Oxygenation

Extracorporeal support for acute respiratory failure was first introduced in the late 1960s and early 1970s.[24-27] ECMO is, typically, high-flow cardiopulmonary bypass modified for long-term patient support. The primary goals of ECMO are an immediate improvement in arterial oxygenation and provision of lung rest to facilitate recovery. In 1974, the National Heart, Lung and Blood Institute sponsored a prospective randomized multicenter trial comparing ECMO to conventional mechanical ventilation in patients with ARDS. The results demonstrated no difference in patient outcome.[27] However, the number of adult ECMO cases has increased steadily in the last 9 years, due in part to the success of neonatal ECMO and the transfer of neonatal skills and knowledge into adult critical care practice. As of July 1995,[28] a total of 197 adult cases had been reported to the International Extracorporeal Life Support Organization (ELSO). This number is dwarfed by the 10,391 neonatal cases reported. Tables 1 and 2 summarize the data on adult cases.

Table 1. Adult ECMO Case Data from the International Extracorporeal Life Support Organization—July 1995[28]

Year on ECMO	Total	Cumulative Total	No. Survived (%)
<1986	2	2	0 (0)
1987	1	3	1(100)
1988	9	12	1 (11)
1989	4	16	2 (50)
1990	18	34	10 (56)
1991	25	59	7 (28)
1992	33	92	14 (42)
1993	28	120	11 (39)
1994	56	176	26 (46)
1995	21	197	8 (38)

Table 2. Adult Cases by Diagnosis—Adult ECMO Case Data from the International Extracorporeal Life Support Organization—July 1995[28]

Primary Diagnosis	Total	No. Survived (%)
Bacterial pneumonia	25	9 (36)
Viral pneumonia	21	14 (67)
Intrapulmonary hemorrhage	1	0 (0)
Aspiration	8	3 (38)
ARDS	52	26 (50)
Other respiratory	32	16 (50)
Pre/post transplant	17	5 (29)
Mitral valve replacement	2	0 (0)
Other cardiac	39	7 (18)

Technical Description

ECMO systems consist of a blood pump, membrane oxygenator for oxygenation and carbon dioxide removal, heat exchanger to warm the blood, and a servo-controlled flow module to regulate pump speed in response to changes in patient venous return. Equipment selection and configuration vary by institution and patient size, as does choice of site for cannula placement. A disposable polyvinyl plastic circuit is primed sequentially with carbon dioxide, crystalloid, albumin, and fresh banked blood. The acid-base status and blood gas content of the blood used to prime the circuit are balanced to that of the patient, with the exception of an elevated partial pressure of oxygen (P_{aO_2}). The circuit blood and the patient's blood are heparinized to prevent clotting. Operative sites are selected and prepared for surgical placement of bypass cannulas. After the cannulas are sutured in place, the venous return and arterial lines of the circuit are attached, using a bubble-free technique. Patient venous blood is drained by gravity into the venous side of the ECMO circuit, where it mixes with the priming blood as it is pumped through the membrane oxygenator (artificial lung) and into the heat exchanger before returning to the patient.

Veno-arterial (V-A) or veno-venous (V-V) routes may be selected, depending upon patient size and need for cardiovascular support. Adult V-A ECMO is recommended for those patients who require cardiac support or interhospital transport on extracorporeal life support (ECLS). Venous blood is drained from the femoral vein and returned to the patient via the right common carotid artery. The advantages of V-A ECMO include (1) provision of full cardiac support; (2) rapid improvement in oxygenation and perfusion; (3) provision for lung rest as consequence of reduction of ventilator settings; and (4) high arterial saturations. Patients who require V-A ECMO for left ventricular (LV) failure must have a left atrial drain placed to decrease LV preload and facilitate ventricular recovery. Disadvantages of V-A ECMO include (1) the need for ligation or repair of carotid artery; (2) the nonpulsatile blood flow that alters perfusion of vital organs; (3) the potential for systemic embolization; (4) the reduction of pulmonary blood flow that may delay lung recovery; (5) the risks of carotid ligation or vascular reconstruction; and (6) the fact that reduction in myocardial oxygen delivery may result in cardiac-stun pathology.[29] (Cardiac stun is a transient loss of ventricular contribution to cardiac output during ECMO and is associated with a higher mortality rate than that seen in ECMO patients without cardiac stun.)

Patients with pure respiratory failure are usually supported with V-V ECMO. Because V-V ECMO provides gas exchange support only, patients with severe hemodynamic instability and left ventricular failure should not be considered for V-V ECMO. Adult V-V ECMO patients are cannulated via the femoral and the right internal jugular veins. This configuration results in mixing of oxygenated circuit blood with deoxygenated patient blood in the right atrium, producing arterial saturations of 80-85%. Increasing ECMO flow in this circumstance does not increase right atrial oxygenation and may increase recirculation of inflow blood. V-V patients have lower systemic oxygen saturations until lung recovery occurs and with it a progressive increase in oxygenation of pulmonary blood flow. Systemic saturations of 80-85% are less well tolerated by adults, and ventilatory variables must be maintained at higher levels than those used in V-A ECMO. The increase in right-atrial oxygen saturations and the pulsatile blood flow present with V-V ECMO may enhance pulmonary parenchymal recovery.[30] The advantages of V-V ECMO include (1) sparing of the carotid artery; (2) preservation of pulsatile blood flow; (3) avoidance of hyperoxia and stunned myocardium; (4) continued perfusion of heart and lungs with oxygenated blood, which may speed recovery; (5) avoidance of systemic embolization. Disadvantages of V-V ECMO include (1) provides no cardiovascular support; (2) prevents accurate assessment of true venous saturation because of partial recirculation in ECMO circuit; (3) limits systemic oxygen saturations; (4) is associated with limited clinical experience as compared to V-A ECMO; (5) is associated with the possible deleterious effects of jugular venous ligation while ipsilateral carotid flow is maintained; and (6) does not facilitate total lung rest because of the need for higher ventilator settings.[30,31] Conversion from one form of ECMO to the other to optimize patient support is possible, but it creates an additional potential for bleeding at the new cannulation sites. Table 3 compares V-A to V-V ECMO.

Figure 1 illustrates a typical adult V-A ECMO circuit. Arrows indicate the flow of blood as it leaves the patient and passes through the ECMO system. Note that the venous cannula (located in the inferior vena cava) has multiple side ports to promote venous drainage. Adults may require two large (4.5 m^2 each) membrane oxygenators connected in series or parallel, as illustrated here, to provide sufficient surface area for gas exchange. Heparin, nutrients, medications, and other liquids can be added to the circuit at the Luer-Lok connections. In-line continuous blood-gas or oxygen-saturation monitors are used to assess the patient (venous limb) and the oxygenator (arterial limb). Pressure monitors are added at various sites in the circuit to increase safety. Pump flow is servo-regulated to the venous return pressure of the patient, in the venous limb of the circuit. The integrity of the oxygenator is assured by maintaining pressure differences across the entry and exit ports of the oxygenator. The blood is warmed to body temperature via the heat exchanger by a countercurrent water bath and then returned to the patient. Figure 2 illustrates a typical adult V-V ECMO circuit. Blood is drained from the superior vena cava and returned via the femoral vein.

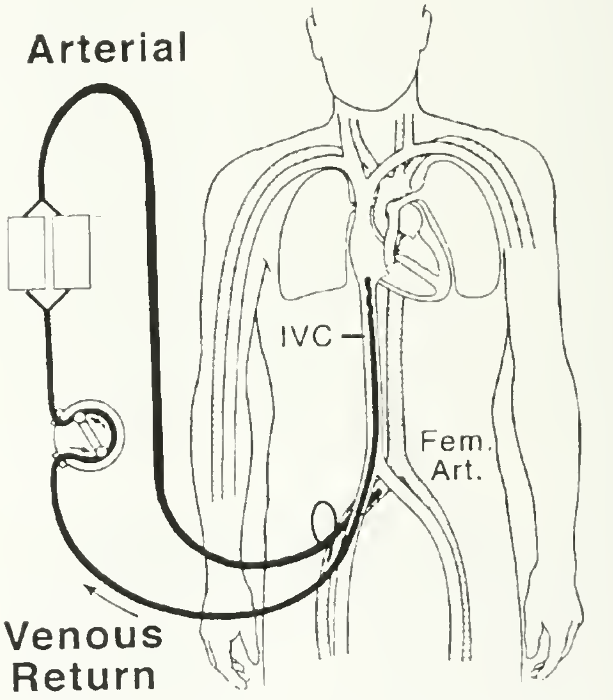

Fig 1. Veno-arterial bypass in an adult utilizing the femoral vein (cannula inserted to the level of the inferior vena cava) as the venous out-flow tract and the femoral artery as the arterial in-flow tract. Note the distal cannulation of the femoral artery to provide blood flow to the leg beyond the cannula insertion site. The adult need for increased surface area for gas exchange is illustrated as two membrane oxygenators connected in parallel.

Table 3 Differences between Veno-Arterial and Veno-Venous ECMO

	Technical			Cardiac		Pulmonary			
ECMO	Ligate Carotid?	Stabilization Rapid	Recirculation?	Cardiac Support?	Cardiac $\dot{D}_{O_2}$?*	Ventilatory Support?	S_{aO_2}	Pulmonary $\dot{D}_{O_2}$?	Pulsatile Blood Flow
V-A	Yes	Yes	No	Yes	Increased	Decreased	Increased	Decreased	No
V-V	No	No	Yes	No	+	< V-A	> V-A	Increased.	Yes

*$\dot{D}_{O_2}$ = oxygen delivery. S_{aO_2} = oxygen saturation.

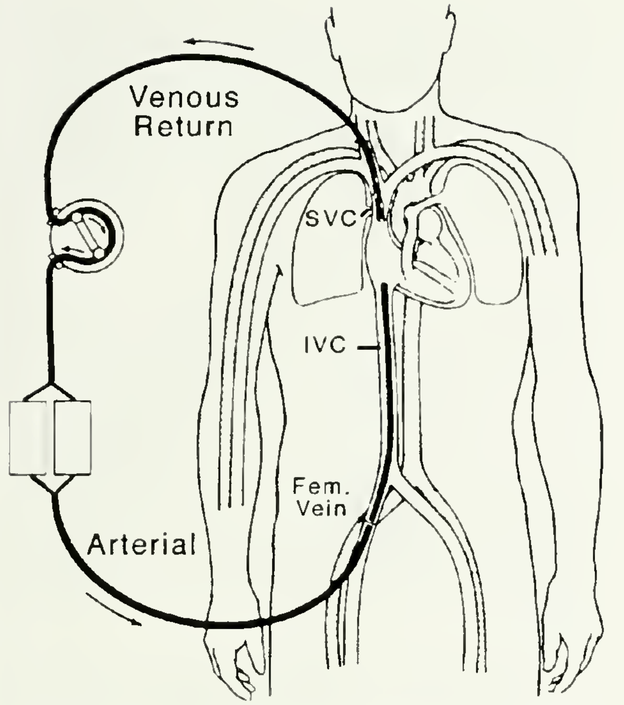

Fig. 2. Veno-venous bypass in an adult utilizing the superior vena cava as the venous outflow tract and the femoral vein (cannula inserted to the level of the inferior vena cava) as the arterial in-flow tract. No distal cannulation is required with this technique.

Clinical Management

ECMO is indicated for patients with reversible, severe respiratory failure unresponsive to maximal medical therapy. Candidates should have reasonable prospects for a meaningful quality of life following ECMO support. The 90% mortality risk level has been suggested as the adult entry criterion for ECMO by the Michigan ECLS group in their Phase-I trial of adult ECMO.[32] The clinical description of patients who have reached the 90% mortality risk group is difficult. Each institution must review its patient population to determine the characteristics of its patients who have a predicted mortality of 90%, given the patient population and the critical care management strategies employed. Table 4 summarizes the Michigan Adult ECMO inclusion and exclusion criteria.[28]

V-A ECMO flow is maintained at 100-150 mL/kg flow or approximately 80% of the predicted cardiac output (C.O.). Oxygen delivery ($\dot{D}_{O_2}$) is a function of the percent bypass received by the patient, arterial oxygen saturation (S_{aO_2}), and hemoglobin oxygen content (C_{aO_2}) of the circuit-patient system ($\dot{D}_{O_2} = C_{aO_2} \times$ C.O.). A lung-rest ventilatory strategy is employed and consists of increased PEEP to maintain end-expiratory lung volume while tidal volume, peak inspiratory pressure, fractional concentration of inspired oxygen (F_{IO_2}), and rate are decreased to reduce risk of ventilator-induced lung injury. The partial pressure of carbon dioxide (P_{CO_2}) in the arterial limb of the ECMO circuit is adjusted by manipulating the gas composition and flow to the membrane oxygenator. During V-V ECMO, flow is maintained at 100 mL/kg. $\dot{D}_{O_2}$ remains dependent upon C.O. and C_{aO_2}; however, manipulations in pump flow may not have as direct an influence on $\dot{D}_{O_2}$. Improvements in tissue oxygenation during V-V ECMO are best achieved by increasing hemoglobin levels and S_{aO_2}.[33] Ventilator settings are adjusted to maintain adequate arterial blood gas values in the patient and are generally higher than those used in V-A ECMO.

Table 4. Selection Criteria for Cardiorespiratory Failure in Adults with 90% or Greater Mortality Risk[28]

Indications
Optimal conventional therapy
Transpulmonary shunt > 30%
Static compliance < 0.5 mL · cm H_2O^{-1} · kg^{-1}
Diffusely abnormal chest x-ray (4-quadrant disease)
(Cardiac failure or cardiac arrest)
Contraindications
Age > 60 years
Duration of ventilation > 5-7 days
Incurable condition
Potential for severe bleeding

A heparin infusion is continuously titrated to maintain activated clotting times (ACTs) from 160-180 seconds[32] to prevent clotting of the ECMO circuit or embolus formation in the patient. Hematocrit and clotting factors are maintained by blood-product transfusion. Coagulation is managed by transfusion of platelets (levels maintained at > 100,000-150,000/mm³) and fresh frozen plasma or cryoprecipitate. Intractable bleeding may be corrected by surgical intervention, with revision of the cannulation site. Nutrients are provided early via total parenteral nutrition and lipid administration to facilitate healing. Patients are initially paralyzed and sedated for surgical or percutaneous cannula placement but then are maintained on sedation levels that facilitate neurologic examinations and patient movement.

ECMO support is reduced as lung recovery occurs and demonstrable improvement occurs in mixed venous oxygen saturation, P_{aCO_2}, hemodynamic status, and urinary output. Trials off of ECMO with the patient's circulation temporarily clamped out of the ECMO circuit assist clinicians in determining the degree of conventional ventilation and inotropic support required to maintain adequate tissue oxygenation. Ventilator modes and settings are examined daily to maximize static compliance and minimize volume overdistention and peak inflation pressures. Therapeutic bronchoscopy may be performed to correct identified areas of lobar collapse. Adults are considered for decannulation when F_{IO_2} is < 0.50, static compliance is 35–40 mL/cm H_2O, P_{aO_2} > 100 torr, and inotropic infusion is < 10 μg/kg/min.[32] Cannulas are surgically removed from the blood vessels, and heparinization is discontinued.

Surgical repair of the cannulated blood vessels may or may not be attempted depending upon the integrity of the insertion site, vessels chosen, and risk for complication.

ECMO complications may be classified as technical or patient related and are summarized in Table 5. Although the equipment and circuit design are simple, technical complications or failures may be life-threatening if lung recovery is not adequate to sustain tissue oxygenation conventionally during a technical repair of the ECMO system. Bleeding, as a result of heparinization, has been reported by Anderson et al[32] to be the most common patient complication, occurring in 88% of their patients. No estimates of daily blood loss were given, but two patients died from intractable bleeding. Organ system failure was the second most commonly reported complication, with renal failure and neurologic complications leading in that category.

Table 5. Complications during Adult ECMO[28]

Patient	Technical (System)
Bleeding—surgical site (35%)	Oxygenator failure (23%)
Bleeding—other hemorrhagic (20%)	Other mechanical (25%)
Organ system failure:	Cannula problems (10%)
Renal—hemofiltration (35%)	
Renal—creatinine $\geq 1.5 < 3.0$ (22%)	
Cardiovascular—inotropes (53%)	
Cardiovascular—arrhythmias (22%)	

Outcomes

The results of extracorporeal support may be defined several ways, ie, improvement in tissue oxygenation, ability to decrease conventional ventilatory requirements, or patient survival. However, morbidity and mortality are the definitive outcome measures by which these techniques should be judged, due to the increased complexity and resource utilization. Survival is commonly defined as discharge to home. The likelihood of recovery as a consequence of the initiation of ECMO support appears directly correlated with length of mechanical ventilation prior to ECMO and with the etiology of the lung disease. Anderson's group[32] initially chose 10 days as the upper limit of pre-ECMO ventilator support. Following preliminary data analysis, they recommended reducing this limit to 5-7 days to improve survival because survival of patients who were mechanically ventilated for longer periods is decreased. The survival data from this Phase-I clinical trial indicate an improved survival rate, as compared to that reported by the National Institute of Health (NIH) ECMO Study Group from the 1970s. Of the Michigan patients, 45% were discharged to home as compared to 9.5% in the NIH study.[27,32] It is important to note that 55% of the Michigan patients recovered from respiratory failure following ECMO support, but only 45% were actually discharged to home. The July 1995 ELSO International Database reports a 38% overall survival for adult ECMO.[28] It may be that the difference in survival between the Database and that reported by Anderson et al[32] can be explained by patient selection and diagnosis. However, the difference in performance between one experienced and resourceful ECMO center and a mix of several centers with varying degrees of experience and success should not be underestimated.

LFPPV-ECCO$_2$R

LFPPV-ECCO$_2$R, or ECCO$_2$R, is a variation of the V-V extracorporeal techniques used during ECMO. Kolobow, Gattinoni, and others pioneered this low-flow V-V bypass technique in Europe.[19,21,34-36] The primary goal of ECCO$_2$R is to remove CO$_2$ via the extracorporeal circuit and membrane lung. LFPPV-ECCO$_2$R is indicated for those adults with severe respiratory failure and hypoxemia when static compliance is < 30 mL/cm H$_2$O, despite optimal positive end-expiratory pressure (PEEP) therapy.[19] Criteria for LFPPV-ECCO$_2$R are listed in Table 6.[19]

Table 6. Criteria for Low-Flow Positive Pressure Ventilation in Combination with Extracorporeal Carbon Dioxide Removal (LFPPV-ECCO$_2$R) Used by Gattinoni and Co-Workers[19]

Inclusion criteria
- Severe hypoxemia
- Static compliance < 30 mL/cm H$_2$O
- Lack of response to PEEP titration (5-15 cm H$_2$O)

Exclusion criteria
- Risk of severe coagulopathy

Technical Description

ECCO$_2$R differs from ECMO in that 70-80% of oxygenation occurs via the patient's natural lungs. The remaining 20-30% support for oxygenation occurs via gas exchange in the extracorporeal circuit. End-expiratory lung volume is maintained by increasing levels of PEEP. Oxygen is administered (1-5 L/min) via a tracheal catheter placed at the carina to replace the quantity of oxygen consumed during apneic oxygenation.[19,21] CO$_2$ clearance is achieved via the membrane oxygenator at a blood flow of 1.5-2.5 L/min and gas flow of 10-20 L/min to the membrane lung. ECMO P_{CO_2} levels are titrated to maintain acceptable patient pH. This may require pump P_{CO_2}s below normal (35-45 torr) because little CO$_2$ removal is performed by the native lung under ECCO$_2$R conditions. Dramatically decreased ventilator support is required with this method, usually consisting of 2-4 sigh breaths/min to prevent atelectasis, with peak inflation pressures limited to 35-45 cm H$_2$O. Plastic circuitry, pump, oxygenator, and heat exchanger are identical to that used for ECMO.

Circulation is achieved using one double-lumen venous cannula, with the outer lumen used for drainage and the inner lumen for return of circuit blood to the patient. The advantages of using a single catheter during ECCO$_2$R are similar to those of V-V ECMO listed previously. The added benefit of single-site cannulation in adults is the ability to perform percutaneous placement, which results in decreased bleeding complications. The disadvantages of this cannulation technique are the slow return of tissue oxygenation and lung function when compared to V-A ECMO. Although oxygenation may improve within 48 hours with this technique, some patients have demonstrated improvement in oxygenation only after 1 to 2 weeks of bypass, with the average length of ECCO$_2$R support reported as 13-19 days.[19,21]

Clinical Management

Heparin management during ECCO$_2$R is similar to management during ECMO, with emphasis on low ACTs (160-180 s) and balanced by coagulation factors administered by transfusion of blood products. Heparin-bonded circuits are also used in this technique; however, these circuits were designed for high-flow bypass, and the low-flow technique used in ECCO$_2$R requires additional heparin to prevent clotting. Nutrition and sedation are also similarly maintained, with early enteral feeding to protect the gut and aid lung recovery and healing.[37] Ventilatory support is decreased as native gas exchange improves. The oxygen concentration is reduced first, using a target P_{aO_2} of 80-100 torr with F_{IO_2} 0.40. Next, the oxygen concentration supplied to the membrane oxygenator is decreased—to 0.21 if possible. If oxygenation remains at the targeted value, end-expiratory pressure is cautiously decreased by no more than 1 cm H_2O every 2 hours. When the ventilator F_{IO_2} is 0.40, membrane F_{IO_2} 0.21, and PEEP 5-10 cm H_2O, spontaneous breathing is allowed using pressure support ventilation or continuous positive airway pressure. At this point, the intratracheal oxygen is also discontinued, and the patient's ability to provide adequate native CO_2 elimination becomes the final criterion to be met prior to decannulation.

Complications are similar to those reported with ECMO; however, there is a dramatic reduction in bleeding with the percutaneous, single-catheter technique.[19] One European study cites transfusion rates as low as 360 ± 1000 mL packed red blood cells/day;[19] whereas, an American ECCO$_2$R study reported transfusion volumes of 2700 mL ± 1900 mL/day.[38] The American study utilized a two-site cannulation technique, further supporting the use of a single double-lumen cannula. Bleeding complications may also be reduced because the double-lumen cannula does not require distal drainage and the sapheno-saphenous percutaneous insertion into the inferior vena cava reduces surgical intervention.

In summary, the advantages that are unique to ECCO$_2$R include (1) decreased bleeding complications; (2) preservation of pulsatile flow to other organ systems; (3) reduction in failure of other organ systems, most likely related to preservation of pulsatile blood flow; and (4) avoidance of systemic embolization. The disadvantages of ECCO$_2$R include (1) slow recovery of lung function; (2) higher requirements for ventilator settings; (3) long recovery period that predisposes the patient to increased risk of technical complications, and (4) requirement for intratracheal oxygen that may complicate ventilator management.

Outcomes

The European ECCO$_2$R group has consistently reported survival statistics of 47%, with fewer complications than experienced with ECMO.[19-21] Several of the technologies that are key factors in ECCO$_2$R success are not available in the United States due to FDA regulations, making comparison of results difficult. Double-lumen catheters, intratracheal oxygen catheters, and heparin-bonded adult circuits are considered experimental and are not widely available. Without these supplies, complications with ECCO$_2$R would most likely mirror those of ECMO.

Length of extracorporeal support for the ECMO groups was 7 days compared to 13-19 days for the ECCO$_2$R group.[19,21,38] Bypass time actually increased in the ECCO$_2$R survivor group between 1986 and 1992, in part because patients were sicker and clinicians had increased confidence in the technique.

A definitive comparison of these techniques can be made only in a randomized, controlled study similar to that performed by Morris et al[38] in which ARDS patients were randomized to one of two groups: protocol-driven conventional ventilator management or pressure-controlled inverse-ratio ventilation with LFPPV-ECCO$_2$R. No significant difference between treatment approaches was seen in this study, with survival reported as 42% and overall patient discharge to home (survival > 30 days) as 38%. Extrapolation to include high-flow ECMO in this comparison must be made informally because entrance criteria may be different even though technique and reported survivals are similar. However, the absence of significant survival differences between the mechanical ventilation and any extracorporeal groups leads to the conclusion that extracorporeal support for acute respiratory failure offers no difference in patient outcome and can be fraught with serious bleeding complications. Table 7 compares the features of ECMO to those of LFPPV-ECCO$_2$R.

IVOX

In 1986, Mortensen[39] reported successful intracorporeal oxygenation in an experimental animal model of extrapulmonary blood gas exchange. This early device utilized intravascular placement of a hollow-fiber oxygenator in the vena cava of the subject. Gas exchange occurred by diffusion as the venous blood contacted the semipermeable membrane on the outer surface of the fibers. In the late 1980s and early 1990s, additional animal studies were reported that updated the development and improvement of the device.[40-42] The term used

Table 7. Comparison of Extracorporeal Membrane Oxygenation (ECMO) to Low-Flow Positive Pressure Ventilation in Combination with Extracorporeal Carbon Dioxide Removal (LFPPV-$ECCO_2R$)

	ECMO[32]	$ECCO_2R$ [38]
Blood flow	High (70-80% C.O.)	Low (30% C.O.)
Type of support	Veno-arterial (cardiac) Veno-venous (respiratory)	Veno-venous (respiratory)
Cannulation	2 sites 2 cannulas (surgical or percutaneous)	Gattinoni: 1 site, double lumen cannula (percutaneous) Morris: 2 sites, 2 single lumen cannula (percutaneous or surgical)
Ventilatory support	Inverse-ratio Pressure-controlled High PEEP 10-12 breaths/min	Pressure-controlled High PEEP 2-4 breaths/min Intratracheal O2
Average length of extracorporeal support	7 - 8 days	13 - 14 days
Cost	NA	Increased variable & contract-adjusted costs[38]
% Survival	45%	47%
Complications	Bleeding (80%): cannulation site & chest tubes Organ failure (renal, CNS) Technical failure	Chest tube bleeding
Causes of death	Termination of support for: Pulmonary artery pressure >75% systemic pressure Irreversible lung disease (pulmonary fibrosis by lung biopsy) Intractable cardiac dysrhythmias	Multiple organ system failure Septic shock Hemorrhagic complications

to describe this technique changed several times during its development—from intravenacaval blood gas exchanger (IVCBGE) to finally being recognized as intravascular oxygenation (IVOX). Phase-I clinical trials were completed in the early 1990s.[43] Table 8 summarizes the entry criteria for this study. A final paper on 164 patients, whose predicted survival from acute respiratory failure was < 20%, was published in 1994[43] and reported that 86% of the patients had some increases in oxygenation with IVOX.

Table 8. Entry Criteria for Clinical Trial of IVOX[38]

Mechanical ventilation for ≥ 24 hours
P_{aO_2} < 60 torr with an F_{IO_2} of ≥
and one of the following ventilator requirements:
    PEEP ≥ 10 cm H_2O
    PIP ≥ 45 cm H_2O
    Mean Airway Pressure ≥ 30 cm H_2O
    P_{aCO_2} ≥ 40 torr with minute ventilation > 150 mL/min/kg

Technical Description

The IVOX device, a small-diameter, elongated, hollow-fiber membrane oxygenator, is inserted into the subject's vena cava. Oxygen enters the device through a catheter and is distributed into the lumina of the hollow-fiber conduits, which lie free in the vena caval blood flowing toward the right atrium. Gas exchange occurs across the selectively permeable membrane comprising the walls of the hollow fibers. The residual CO_2 within the lumina of the fibers is then discharged through the double-lumen catheter.

The catheter is surgically inserted into the right femoral vein below the inguinal ligament. Figure 3 demonstrates placement of the unfurled fibers in the vena cava.[20] A tubular sheath is placed antegrade into the vein. The IVOX device is inserted through the sheath and advanced until the tip lies in the superior vena cava. Placement in the venous system provides the added benefit of having the lungs to filter and protect the brain from emboli, if such are formed. Gas exchange is proportional to the membrane surface area and the diameter of the common femoral vein at the entrance site in the groin. In theory, a 9,300 cm^2 adult device with 4 L of blood (cardiac output) flowing over it would transfer 157.7 mL of oxygen and 224.6 mL of CO_2/min.[44] An oxygen line is attached to the inlet limb of the catheter, and gas flows through the device from a precisely calibrated, adjustable regulator. The exit limb has low negative pressure applied to it to facilitate removal of CO_2 and promote gas flow through the device. The flow of gas is adjusted to maintain targeted blood gas values. Pressure of gas within the hollow fibers is maintained at less than the central venous pressure to reduce the risk of gas embolism. Figure 3 demonstrates placement of the IVOX device in the vena cava.

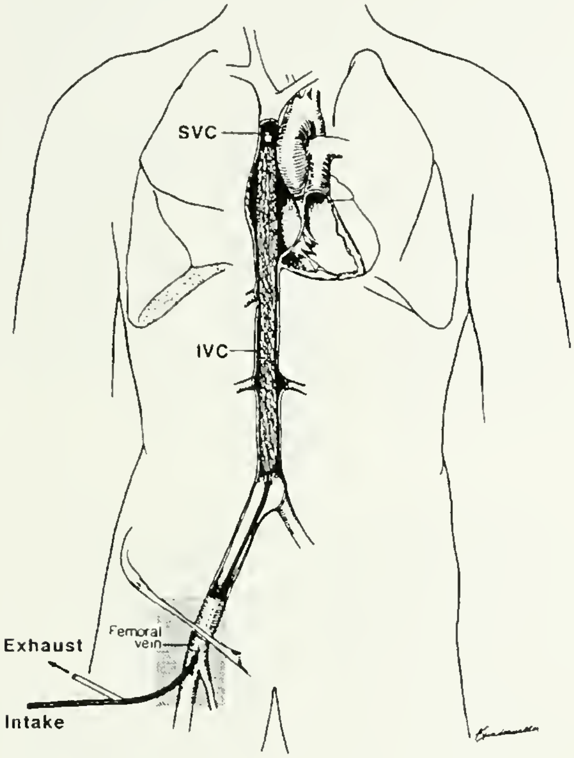

Fig. 3. Placement of the unfurled IVOX device in the vena cava. The upper tip of the device rests in the superior vena cava at the level of the jugular vein and extends through the inferior vena cava. Note the gas intake port and the exhaust port, which lie outside the skin.

Clinical Management

Systemic heparinization is required prior to insertion and is continued to prevent clotting and blood emboli formation. ACTs are maintained at 180-200 seconds throughout implantation.[43] Anticoagulation and bleeding are managed in a similar fashion to that discussed for ECMO, with emphasis on short ACTs and on providing a balance of coagulation factors via transfusion of blood products. As oxygenation improves, intensity of mechanical ventilatory support is progressively decreased. Catheters have been kept in place for up to 18 days in some human subjects.[43] Complications related to the use of IVOX have been classified using a 4-step severity scale, ranging from "not clinically or pathologically important" to being a "primary or contributing cause of death."[43] Of the adverse events observed, 39 were classified as moderate in severity, and 7 were rated as severe, indicating that they were probably related to the patient's death. Bleeding was the most commonly reported complication, with insertion and removal of the device being the primary causes. Venous thrombosis and thrombophlebitis were the next most common complications. No hematologic abnormalities or toxic, allergic, or rejection responses were observed by the investigators. Of the IVOX devices used, 29.3% had mechanical or performance problems, with the leak of blood into the hollow fibers being the most common. The device was removed from 12 patients for this reason. Gas transfer insufficient to meet patient metabolic needs was reported in 39 cases and is borne out by the lower than predicted CO_2 and O_2 transfer rates reported (Table 9).[44]

Table 9. Average Quantitative Gas Transfer Rates Achieved by IVOX during Clinical Trials[44]

Size of IVOX	CO_2 Transfer (mL/min)	O_2 Transfer (mL/min)
7	40.3	43.8
8	45.6	60.2
9	54.2	60.1
10	72.5	71.0

Outcomes

Measurable improvements in P_{aO_2} and P_{aCO_2} were observed within the first few hours following implantation in most patients. Reduction of mechanical ventilation by at least 25% of pre-IVOX levels was achieved in 50% of the patients in the trial.[43] However, statistical significance was not reported for these reductions. The IVOX-treated patients had a 30% hospital survival. Patients with the lowest lung-injury scores (least severe acute respiratory failure) had the best survival rates, with the Murray Lung Injury Score correlating well with these results. Clinical investigators were pleased with the simplicity and convenience of the IVOX device but were disappointed that the device could not transfer more O_2 and CO_2.[43,44] For these reasons, a controlled Phase-II and Phase-III study was never conducted. The IVOX device remains experimental, with no clinical trials underway (1995).

Cost Issues

The cost and efficacy of high technology procedures like extracorporeal and intracorporeal support require careful scrutiny prior to endorsement. Results of Phase-I trials, which demonstrate improvements in physiologic variables must be followed by Phase-II and III trials that compare conventional techniques and examine outcome versus cost issues. Cost data for extracorporeal support are difficult to delineate because costs vary by country, institution, and standard of practice. Intracorporeal cost data are nonexistent because these devices were available only under sponsored research protocols and were never commercially distributed.

Several neonatal ECMO cost studies have been conducted to examine the cost and efficacy of extracorporeal support versus traditional therapies.[45-48] A retrospective study conducted at Children's National Medical Center in 1986[46] compared the cost of ECMO therapy with the cost of conventional care for a similar clinical population. The average length of stay for ECMO patients (25 days) was significantly shorter than for the non-ECMO group (75.8 days, $p < 0.005$). The decrease in hospital length of stay, despite the increased ECMO cost, resulted in a 43% reduction in total physician and hospital charges. The survival for the ECMO-treated group quadrupled. These data have been supported by two similar studies.[47,48] This is powerful information to support the use of ECMO in neonates, but the cost-benefit data for adult extracorporeal support does not appear as positive. Morris et al[38] reported cost and survival data in the Utah study of LFPPV-ECCO$_2$R versus conventional mechanical ventilation. Total charges and survival for each group are summarized in Table 10. Survivors in the ECCO$_2$R and conventional groups had significantly higher charges than nonsurvivors, presumably due to the increased length of hospital stay. Variable and contract-adjusted charges for the ECCO$_2$R group were significantly higher than for the conventional group, with no significant difference in survival. Cost data from a similar control group for adult ECMO has not been published. Conclusions based on the limited available data are difficult, but the failure to significantly reduce cost or improve survival using extracorporeal techniques in adults is not encouraging.

In Summary

Most investigations of extracorporeal and intracorporeal support for acute respiratory failure have not progressed beyond Phase-I safety trials in adults. The lack of standardization and reproducibility in equipment, technique, length of extracorporeal support, and rate of complications further complicate the assessment of these techniques and limits the ability to make strong clinical recommendations. Without control group data, institutional baselines for conventional management outcomes and costs are unknown, and valid comparisons cannot be made.

Based on this information, recommendations can be made. Multicenter, prospective, randomized, controlled clinical trials utilizing all available modern techniques and carefully developed treatment protocols must be conducted to conclusively answer the efficacy and cost-benefit questions for each of these technologies. Until the techniques are standardized and these studies can be conducted, extracorporeal and intracorporeal techniques for support of these patients must be considered experimental. Because of the limited data and experience with these procedures, clinical trials should be permitted only in those research centers with documented experience and active controlled clinical trials. Research to advance knowledge and skills related to mechanical ventilation must continue, as early data from these groups suggest improved morbidity and mortality. Conventional mechanical ventilation currently demonstrates the best cost-benefit profile for the patient and medical institution.

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Table 10. Clinical Costs and Charges from LDS Hospital Admission to Death or Discharge in Thousands of Dollars (Physician Cost & Charges Excluded)[38]

		Daily Costs*		
	Number	Fixed	Variable	Adjusted Contract
Control	19	1.59 (0.09)	1.69 (0.12)	4.11 (0.12)
Live	8	1.18 (0.06)	1.15 (0.04)	2.92 (0.12)[†]
Die	11	1.89 (0.08)	2.08 (0.11)	4.97 (0.23)
New	21	2.25 (0.24)	2.44 (0.27)	5.87 (0.63)
Live	7	1.33 (0.08)[†]	1.40 (0.08)[§]	3.42 (0.19)[‡§]
Die	14	2.71 (0.3)	2.96 (0.32)	7.09 (0.76)

*Costs are mean (SEM) values in thousands of dollars; [†] = significantly different from cost in control group who died; [‡] = significantly different from new therapy patients who died; [§] = significantly different from control patient who lived (Mann-Whitney $p < 0.001$).

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Wilson Discussion

East: To clarify some issues with you—In the LDS $ECCO_2R$ trial, I think they went far out of their way to try to duplicate the European methods. They sent a team who assisted in running $ECCO_2R$ over there. The circuit they put together was actually identical to the one used by Gattinoni when he published his 70% survival.[1] Now, admittedly, they've gone to the fancier dual-lumen catheters, but when they started the trial, it was an exact duplicate of Gattinoni's system—even down to the pump and the circuits and everything. Then they brought two experienced physicians over from Milan to Salt Lake, and they are the ones who started running this trial. Everybody says "You guys just don't know how to do $ECCO_2R$." I think they really tried hard to get the method transferred. If you can't transfer it with that kind of expense and effort, you wonder if *anybody* can learn it. The physicians who visited in Salt Lake said that they would never pump these patients in Milan. The issue is entry criteria. You present survival rates as if they're all comparable, but the survival rates from the European literature[2] were from studies that had no rigid entry criteria. They're all sick patients, clearly. But what does a 47% survival rate mean when you include some patients who were too sick to meet the rigid entry criteria that Alan (Morris) used,[3] which were the old NIH ECMO entry criteria. The general feeling of the physicians from Milan who worked with us was that those patients are 'dead ducks.' They wouldn't pump them in Milan. So, you may be seeing what you alluded to—you have to pick these patients carefully. Then you wonder, Would they do just as well on the 'vent' later? The other issue is cost. We must make the distinction that what was reported are actual costs during the trial, which do not include the pump technician, the circuits, or the extra diagnostics and blood products.

1. Gattinoni L, Pesenti A, Mascheroni D, Marcolin R, Rumagalli R, Rossi F, et al. Low-frequency positive-pressure ventilation with extracorporeal CO_2 removal in severe acute respiratory failure. JAMA 1986;256(17):881-886.
2. Lewandoski K, Slama K, Falke K. Approaches to improved survival in ARDS. In: Vincent JL, editor. Yearbook of intensive care and emergency medicine. Berlin: Springer-Verlag, 1992.
3. Morris A, Wallace C, Menlove R, Clemmer TP, Orme JF Jr, Weaver LK, et al. Randomized clinical trial of pressure-controlled inverse ratio ventilation and extracorporeal CO_2 removal for adult respiratory distress syndrome. Am J Respir Crit Care Med 1994;149(2, Part 1):295-305. *See the published erratum Am J Respir Crit Care Med 1994;149(3, Part 1):838.*

Wilson: I did not include the physician's cost or any of those charges in the data that I presented.

East: So if you were using it clinically, the difference would be much greater than what Gattinoni reported. I understand that some ongoing work has shown that you can get better CO_2 removal if you use permissive hypercapnia and IVOX at the same time. I think there are groups that are continuing to try to develop better fibers.

Wilson: When I talked to Jay Zwischenberger about the technique a couple of weeks ago, he confirmed that. Two groups are still actively pursuing the IVOX technique. There's one in Chicago, and I believe there's one in New England and one in Pittsburgh. It makes sense that the patients with permissive hypercapnia have a better response with IVOX because gas exchange is pressure-gradient driven. The patients in the IVOX trial who had the best gas exchange were the most hypoxic because the partial pressure difference was greater. I agree with everything you said, Tom, and I'm glad that you spoke to the Utah experience with $ECCO_2R$. I think that they did do an incredible job in trying to duplicate the technique. I think the take-home message is that it is impossible to duplicate some of these techniques and outcomes from center to center. It appears that it is the unique combination of individuals, equipment, and experience that accounts for different survival rates. I did not mean to imply that the patient survival rates I reported are directly comparable. My intention was to offer some published information that would allow us to draw some conclusions about these techniques.

East: Yes, but I think you have to be careful when you're drawing the conclusion that some centers do the technique better. I'm not convinced of that. I think some centers report better outcomes because of their patient selection.

Wilson: That's true. We could see that if we were able to compare the patient-selection criteria. I believe you're absolutely right. I also think, though, that center-specific techniques may produce different outcomes.

Hurst: If ever there were techniques that cried out for regionalization, we've just heard about them—not only because of the experience issues, but also because of the up-front resources that have to be committed. If we're ever to be able to realize the true value of these techniques in our armamentarium, certain (not all) centers are going to have to be really dedicated, as you are, to providing them. Certainly, we who treat adults will never be able to approach, nor should we think we can approach, the outcomes that you can achieve in the pediatric age group. The pathology is too varied. When you look at all the complicating factors, particularly in the trauma patient, for example, the bleeding issues are monumental. 45% of those patients may have a serious intracranial injury and over 50% of those patients may have a serious orthopedic injury. Many are complex pelvic fractures. Some of those patients may have undergone complex hepatorrhaphies. We would not want to submit any of those patients to systemic anticoagulation. We would like to, but, unfortunately, we simply can't. I think before we try to compare one group to another, we need to agree that there's a physiologic scoring system that will allow us precise comparison, so we know what those survival rates really mean. The reason we have shied away from the carotid artery ligation techniques goes back before the modern age of vascular surgery, when the treatment of choice for carotid injuries was indeed carotid artery ligation.[1] That procedure was accompanied by a major neurologic sequelae rate in excess of 20%. So, we get rather nervous when you mention ligating the carotid artery in the adult.

1. Rich NM, Spencer FC, editors. Carotid and vertebral artery injuries. In: Vascular trauma. Philadelphia: WB Saunders, 1978.

Peruzzi: You're correct about the bleeding and anticoagulation is a problem. We had a lot of bleeding complications. I had occasion to speak with an investigator from one center that entered a relatively large number of patients in the IVOX protocol but did not report many bleeding complications, but they were using the activated clotting times (ACTs) to follow anticoagulation status. When they actually went back and looked at the prothrombin times (PTTs) obtained at the same time that they were getting the ACTs, they found that they really were not anticoagulating the patients to the extent that they had thought that they were, guided by their ACT measurements. We all believed that the IVOX did not really require the anticoagulation that was dictated by the protocol, and probably could have gotten away with much less or none. As a matter of fact, they were doing a lot with heparin bonding, and I think that probably would have been adequate. But the bottom line is we had to follow the protocol, and that's where we found the difficulties. Gas exchange is always going to be an issue. Gradients? Yes, gas exchange improves if you increase the gradient, but, by and large, we were never really able to seriously decrease the level of support by more than 20% or so. The one thing that I will say in favor of the IVOX device is that it was extremely user-friendly. Once one got it in place, it really was pretty unobtrusive. The bedside nurses handled things very well. The pump ran, and it didn't require a lot of user support, as do ECMO and $ECCO_2R$. That was an advantage. Unfortunately, as you say, the gas-transfer limitation is the biggest problem.

Wilson: I think your comment about the bleeding is right, Bill. The bleeding really needs to be thought of in two contexts with the IVOX device—bleeding at the site of insertion and leakage of blood into the gas path. Implantation caused the most bleeding. At this stage, 69% of patients lost 100-500 mL of blood. Leakage into the gas path was secondary to concern for bleeding at the insertion site.

Peruzzi: From our experience, the leakage into the gas path wasn't that bad. It depended on the insertion. If the fibers broke during insertion you got some blood in there, but we didn't notice that problem much. We had people bleed into their chests: we lost a good deal of blood during insertion of the device, and we had all kinds of other extraneous bleeding problems, but our experience was different than what you just described.

Wilson: I think if I recall from the Conrad paper,[1] 22 of 164 (13%) of cases bled into the gas path. And you're right—good insertion technique is very important to prevent fiber breakage and bleeding.

1. Conrad SA, Bagley A, Bagley B, Schaap RN. Major findings from the clinical trials of the intravascular oxygenator. Artif Organs 1994;18(11):846-863.

Mathews: Do you have material on long-term sequelae in those people who were discharged? Did they have neurologic deficits? Physical disabilities? Decreased quality of life, like we see in the kids?

Wilson: Obviously the neonatal outcome data are the best data. There were 197 total adult cases in the ELSO registry, compared to 10,000 neonatal cases, so there's not a lot of information on adult follow-up. I believe in the University of Michigan group, of the patients who survived and were discharged home, 21 returned to normal functioning, in terms of jobs and lifestyles. One patient had other complications that prevented full recovery. So, long-term follow-up data are not readily available, and we have only a very, very small number of patients.

Mathews: In at least one of the neonatal studies that I read, approximately 24 of 119 survivors showed some kind of neurologic (n = 14) or developmental defect (n = 14). If early death (n = 27) was included, the percentage at 1 year increased to 46%.[1]

1. Graziani LJ, Streletz LJ, Baumgart S, Cullen J, McKee LM. Predictive value of neonatal electroencephalograms during extracorporeal membrane oxygenation. J Pediatr 1994;125(6, Part 1):969-975.

Wilson: The problem with that, Paul, is that you don't know when that injury occurred—whether it was the severe hypoxia prior to extracorporeal support or as a result of the extracorporeal support. So that's why when they assess follow-up data they try to look at controls of patients who had some kind of similar conventional therapy and disease severity scores.

Minimizing Alveolar Stretch Injury during Mechanical Ventilation

Neil R MacIntyre MD

Normal Alveolar Distention
Stretch Injury & Alveolar Overdistention
Measuring Alveolar Distention & Setting Ventilatory Targets
Strategies To Minimize Alveolar Overdistention
    Minimizing PEEP
    Minimizing V_T
In Conclusion

Normal Alveolar Distention

The normal, healthy lung is inflated and deflated as changes in transpulmonary pressure are generated by the inspiratory muscles (Fig. 1[1]). The normal end-expiratory volume (functional residual capacity, or FRC, Fig. 1) is reached when ventilatory muscles are relaxed and transpulmonary pressures are zero. The normal maximal lung volume (total lung capacity, or TLC, Fig. 1) is reached when ventilatory muscles generate transpulmonary pressures of 30-40 cm H_2O. Thus, during spontaneous breathing, the lung is never subjected to distention from transpulmonary pressures exceeding 30-40 cm H_2O (absolute value). However, this may not be the case during positive pressure ventilation.

Stretch Injury & Alveolar Overdistention

Common approaches to mechanical ventilation involve periodic inflations of the lung using positive airway pressure. Conventional strategies utilize tidal volumes (V_Ts) of 8-12 mL/kg, respiratory rates of 10-30 breaths/min, supplemental oxygen, and levels of positive end-expiratory pressure (PEEP) to stabilize atelectatic lung units.[2,3] In general, these strategies are designed to mimic the normal ventilatory pattern. However, gas exchange needs and other considerations have, in the past, prompted application of inflation pressure often much higher

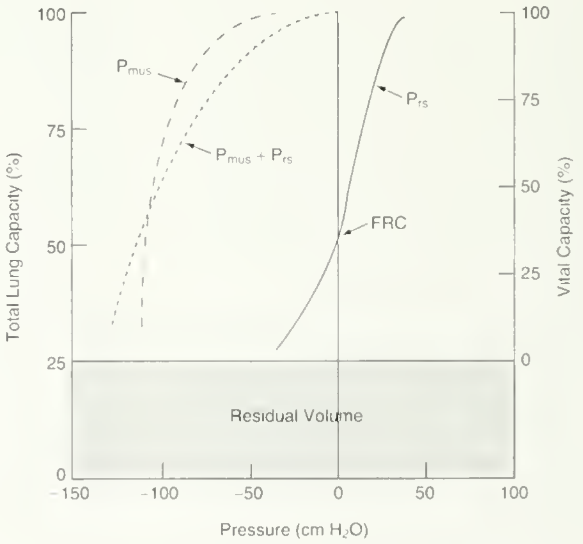

Fig. 1. Normal respiratory system mechanics during inspiration for a spontaneously breathing patient. Lung volume is plotted on the vertical axis, various pressures are plotted on the horizontal axis. Respiratory system compliance is represented by volume changes as a function of transrespiratory system pressure (P_{rs}, solid line). Pressure generation capabilities of the inspiratory muscles over the range of inflation is P_{mus} (dashed line). Functional residual capacity (FRC) is the relaxation point of the respiratory system when transrespiratory system pressure is zero at end exhalation. Inflation is possible when P_{mus} is more negative than P_{rs}, ie, the sum of $P_{mus} + P_{rs}$ is negative (dotted line). Total lung capacity thus occurs when the absolute value of P_{mus} equals that of P_{rs}. As can be seen, this maximal voluntary inflation point occurs when P_{mus} –30 to 40 cm H_2O and P_{rs} is +30 to 40 cm H_2O. (Adapted from Reference 1, with permission).

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A version of this paper was presented by Dr MacIntyre during the RESPIRATORY CARE Journal Conference, Mechanical Ventilation: Ventilatory Techniques, Pharmacology, & Patient Management Strategies, held in Cancún, Mexico, October 6-8, 1995.

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than the normal maximal transpulmonary pressure. Under these conditions, lung units may become distended beyond their normal capacity and, thus, sustain injury.

The potential for lung injury because of positive pressure breaths has long been appreciated. The best known form of injury occurs when positive pressure breaths grossly overinflate the lungs and result in air leaks, such as pneumothorax, pneumomediastinum, and subcutaneous emphysema.[2-5] The mechanism for this type of injury is thought to be actual alveolar rupture into the perivascular space with subsequent dissection of air into the mediastinum, pleura, and other locations.[4-6] The risk of alveolar rupture is high when transalveolar pressures approach 50-60 cm H_2O.

In recent years, data have shown that alveolar distention resulting from transpulmonary pressures only marginally above the maximum normal level of 30-40 cm H_2O may also be associated with lung injury.[7-20] This injury, however, does not involve alveolar rupture but is characterized by a tissue injury involving the alveolar-capillary interface. The presumed mechanism is that alveolar stretch beyond its maximum normal level disrupts the alveolocapillary membrane. This causes parenchymal inflammation, capillary fracture,[21] increased vascular permeability, abnormal accumulation of lung water, abnormal surfactant function,[22] alveolar flooding, atelectasis, infiltrates visible on radiograph, and hypoxemia because of shunting of blood. Injuries of this type may be most pronounced in lung units that were previously healthy. This is because lung injury is not always distributed uniformly (ie, some lung units are severely injured and atelectatic while other units remain relatively normal),[23,24] and, thus, positive pressure ventilation delivered to the airway may only minimally inflate abnormal units but may overdistend and consequently injure the healthier units (Fig. 2). Therefore, conventional mechanical ventilation goals to aggressively recruit and ventilate abnormal lung units with distending pressures in excess of 30-40 cm H_2O may risk overdistending (stretching) and injuring the remaining healthy lung units.

Most of the evidence for this stretch injury comes from animal studies (Table 1). From these studies, it appears that *maximal* stretch is the key component of injury although increasing *duration* and *frequency* of stretch may further increase injury.[10,18,19] The role of PEEP in the development of stretch injury is more complex.[7,20,25-27] On the one hand, if PEEP elevates FRC primarily by distending already patent alveoli, further increases in PEEP for a given V_T increases maximal end-inspiratory alveolar stretching. On the other hand, PEEP may serve to increase FRC through recruitment of collapsed alveoli. This may affect stretch injury differently because alveolar collapse during expiration requires the next positive pressure breath to 'snap open' the alveolus before it can fill with gas.[25,26] This repetitive collapse and snapping open appears to contribute to the parenchymal injury associated with positive pressure breathing. Indeed, a number of animal studies have shown that providing enough PEEP to prevent collapse (ie, maintaining alveolar recruitment) significantly reduces the incidence of pulmonary edema, hyaline membrane formation, and other conditions known to contribute to alveolar injury.[7,20,25-27]

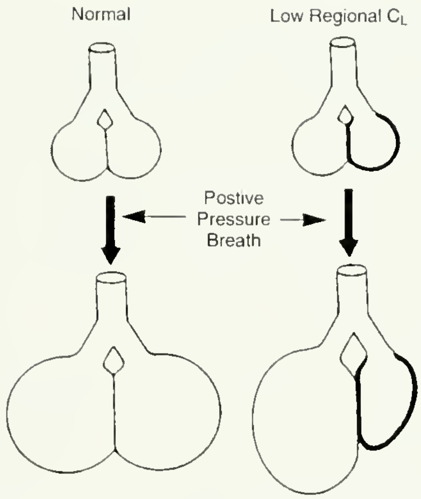

Fig. 2. The volume from a positive pressure breath distributes homogeneously throughout a lung with normal compliance (C_L) (left panel). In a lung with low regional C_L, the volume from a positive pressure breath distributes preferentially to the regions with more normal C_L (right panel). Thus, a V_T of normal size in a lung with regions of low C_L can overdistend the healthier regions.

Moreover, the studies listed in Table 1 also show that PEEP reduces lung injuries resulting from overdistention.[7,10,15,17-20] Additional support for the lung-protection association with PEEP is found in rabbits during high-frequency oscillatory ventilation, in which maintenance of an adequate baseline lung volume was associated with fewer and less severe histologic abnormalities in injured lungs.[30] There are much fewer human data on this subject, but neonates on extracorporeal systems (ECMO) have been shown to have their need for ECMO shortened if expiratory pressure is provided.[31] Taken together, these data suggest that although PEEP can increase maximal end-inspiratory alveolar distention (and thus *increase* the potential for stretch injury), a certain minimal PEEP to prevent collapse may *decrease* the stretch injury.

Because pathologic findings related to stretch injury are similar to those observed in acute lung injury (ALI) and acute respiratory distress syndrome (ARDS), it may be that lung injury from overdistention exacerbates or prevents resolution of ARDS. Therefore, recovery from respiratory failure and ultimate survival might improve if ventilation strategies were used that avoid or reduce the application of stretching forces to the lung. Indeed, this is the conclusion of an uncontrolled trial of low-stretch ventilation[28] and of a recent preliminary report of another clinical trial.[29] It is also the reason that a recent consensus recommended stretch-induced lung injury be reduced by avoiding high ventilating pressures.[2]

Table 1. Results of Studies on Pressure-Related Lung Injury

First Author	Model	Lung Distention Pressures (cm H_2O) P_{peak}*	PEEP	$\bar{P}_{aw}$	Lung Injury
Webb[7]	Rats	14	0	3.6	1+
		30	0	6.7	2.5+
		45	0	9.7	3.5+
		30	10	14.2	2+
		45	10	17.8	3+
Parker[15,17]	Dogs	60	0		2+
		50	0		1.5+
		40	0		1+
		30	0		0
		15	0		0
Tsuno[18]	Sheep	15	4		1+
		30	4 (rate = 4)		2+
		30	4 (rate = 15)		3+
Kolobow[10]	Sheep	20	7		1+
		50	7 (rate = 3)		3+
		50	7 (rate = 12)		4+
Tsuno[19]	Pigs	18	4		0
		40	4		1.5
Dreyfuss[20]	Rats	8	0		0
		15	0		0
		45	0		3+
		22	10		0
		45	10		1.5+
		37	15		1+

*P_{peak} = peak airway pressure, PEEP = positive end-expiratory pressure, $\bar{P}_{aw}$ = mean airway pressure, rate = ventilator cycling frequency, lung injury = relative score with 1 being normal and 4+ being most severe

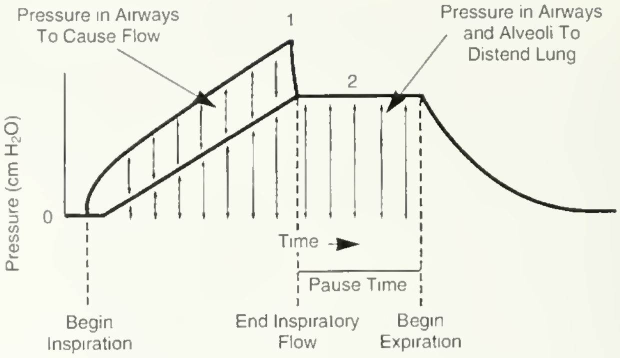

Fig. 3. Airway pressure during a positive-pressure breath. During gas delivery, pressure initiates flow and distends alveoli (peak pressure, Point 1). If inspiration is held (paused) after flow has ceased, airway pressure plateaus and represents only alveolar distending pressure (plateau pressure, Point 2).

Measuring Alveolar Distention & Setting Ventilatory Targets

Measuring and monitoring alveolar volume is difficult. However, approaches that assess pressures or the pressure-volume relationship can be taken to indirectly assess the risk of stretch injury and guide strategies to manage it.

The simplest measurement is the so-called airway plateau pressure (Fig. 3). This is the airway pressure at end inspiration under no-flow conditions and, as such, reflects alveolar pressure. Assuming the patient-ventilator system is synchronous, the compliance of the chest wall is normal, and abdominal pressure is low, plateau pressure is a reasonable reflection of maximal transalveolar pressure and can be used to estimate the collective distention of lung units of varying compliance. Keeping plateau pressures below 30-40 cm H_2O to prevent normally compliant units from exceeding their maximum volume has been recommended (Fig. 1).[2]

A more sophisticated approach that assesses both alveolar overdistention and recruitment is the static pressure-volume (P-V) plot (Fig. 4).[32-34] When properly done, this plot can be used to detect both a lower and an upper inflection point (P_{flex}), which can be used to detect changes in the lung's pressure-volume relationship. The lower P_{flex} has been described as the level above which PEEP should be set to avoid alveolar collapse and snap-open injury. The upper P_{flex} has been described as the level above which the application of additional pressure results in only a small volume increase and is associated with overdistention. However, plotting a static P-V curve is a cumbersome procedure requiring multiple breaths of varying size and, often, patient sedation or paralysis. Thus, it is not a practical bedside procedure.

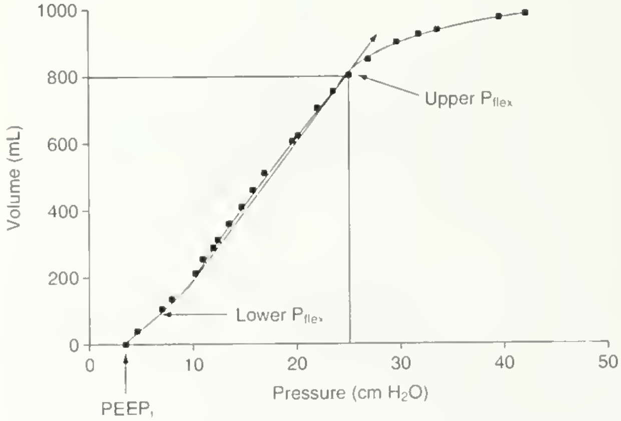

Fig. 4. Static inspiratory pressure-volume plot in a patient with ARDS. In this example, 3 cm H_2O intrinsic PEEP ($PEEP_i$) is present. Lower and upper inflection points (P_{flex}) can be seen. These are believed to represent minimal recruitment pressures (lower P_{flex}) and overdistending pressures (upper P_{flex}). The ideal ventilatory pattern using these measurements would be a PEEP just above the lower P_{flex} and a V_T-PEEP combination that prevented peak airway pressures from exceeding the upper P_{flex} (Reprinted from Reference 32, with permission).

A promising and clinically applicable approach has been proposed that utilizes a constant-flow mechanical breath at varying levels of PEEP to identify both the minimum level of PEEP required to maintain alveolar recruitment and the overdistention pressure.[33]

Strategies To Minimize Alveolar Overdistention

Reducing alveolar overdistention during mechanical ventilation can be accomplished by maintaining either baseline pressure and volume at minimum levels or tidal pressures and volume distention at minimum levels. However, concessions may be required in terms of blood-gas values (Table 2).

Table 2. Concessions To Consider When Minimizing Positive End-Expiratory Pressure (PEEP) and Tidal Volume (V_T)

Minimizing PEEP	
Clinical Targets:	Reduction of baseline volume and pressure to prevent or decrease alveolar overdistention with the applied tidal breath
Concessions:	Requirement for higher F_{IO_2} because of lower S_{aO_2}, increased risk of alveolar collapse, and risk of ventilator-induced injury because of snap-open phenomenon if PEEP is set below the lower inflection point
Minimizing V_T	
Clinical Targets:	Reduction of V_T and peak inspiratory pressure to prevent alveolar overdistention
Concessions:	Acceptance of higher P_{aCO_2}, lower pH, lower S_{aO_2} and need for higher F_{IO_2}

Minimizing PEEP

PEEP is the baseline pressure applied to patients who are intubated and mechanically ventilated; it establishes and maintains end-expiratory or baseline lung volume—FRC. Minimizing PEEP, however, requires concessions. On the one hand, a certain level of PEEP may be needed to prevent alveolar collapse and reduce lung injury due to the snap-open phenomenon. Moreover, alveoli that do not collapse provide better gas exchange and, thus, allow use of a lower F_{IO_2}. On the other hand, high PEEP, applied in an attempt to normalize P_{aO_2}, may be counterproductive if it results in alveolar overdistention.

Practical ways to assess adequate, yet safe, PEEP and peak airway pressure are needed. In the absence of P-V plots, one approach is the use of gas-exchange variables. For example, clinical studies have demonstrated that the lower P_{flex} is generally in the range of 5-15 cm H_2O in patients with ARDS who require $F_{IO_2} > 0.40$.[29,32-34] Thus, providing a minimum level PEEP of 5-15 cm H_2O in these patients may be prudent. Another approach involves setting "best" PEEP based on the compliance of the respiratory system (C_{rs}). Because the slope of the linear, rising portion of the P-V curve between the lower P_{flex} and the upper P_{flex} reflects C_{rs},[35] the magnitude of this variable derived from a tidal breath may be a reasonable surrogate for plotting the two inflection points. With this approach, a tidal breath approaching maximum normal distention (ie, 25-30 cm H_2O) with PEEP of 15 cm H_2O might be used to initially recruit all recruitable alveoli. The best PEEP might then be determined by measuring C_{rs} as PEEP is progressively lowered to a minimum of 5 cm H_2O. Best PEEP, then, is that which results in the highest value of C_{rs}.

Because a level of PEEP set by mechanics measurements may not result in maximum values of P_{aO_2}, this strategy may require a higher F_{IO_2}. Alternatively, arterial oxyhemoglobin saturation (S_{aO_2}) lower than that usually considered acceptable may be tolerated (eg, 85-88%) provided oxygen delivery can be maintained using other methods, such as increasing cardiac output and/or increasing hemoglobin concentration.[2] Prolonging inspiratory time can also be used as an alternative to additional PEEP, when alveolar recruitment seems optimal but oxygenation is inadequate. A longer inspiratory time has two effects. First, there is a longer gas-mixing period within the alveoli, and the time of exposure of capillaries to gas-filled alveoli is extended. This alone may serve to reduce the alveolar-arterial oxygen difference.[37] A second effect of a prolonged inspiratory time is the prevention of complete lung emptying because of the consequently shorter expiratory time that may produce intrinsic PEEP and result in air trapping.[38] It is well known that intrinsic PEEP functions much like applied PEEP in its effects on FRC.[38] However, the effects of intrinsic PEEP may be primarily within easily distended lung units, placing them at special risk for rupture.[39] Thus, alveolar overdistention can again become a problem when air trapping and intrinsic PEEP develop and prolonged inspiratory time must be used with caution and with careful monitoring of intrinsic PEEP.

Minimizing V_T

Alveolar distention can be minimized by limiting tidal-volume distention. In the past, delivered V_Ts as high as 15-20 mL/kg were often recommended. This recommendation evolved from the practice in anesthesia before the development of PEEP, when large V_Ts were required to overcome atelectasis. Lowering V_Ts to 6-8 mL/kg reduces alveolar pressures and alveolar distention.[28,29,40] To some degree, alveolar ventilation can be maintained by increasing the respiratory frequency. Ultimately, however, rapid rates cannot compensate for the loss of V_T. During ventilation with low V_T, alveolar ventilation falls, arterial P_{aCO_2} rises and arterial pH falls.[2,28,29,41,42] This strategy is often referred to as permissive hypercapnia, and pHs with this strategy have been reported to fall below 7.0. The effects of normoxic hypercapnia are only beginning to be understood. In general, however, human subjects appear to tolerate an arterial pH as low as 7.15 and P_{aCO_2} of 80 torr quite well.[41] The rate at which the P_{aCO_2} is allowed to increase should be slow (eg, 10 torr/h) to allow time for intracellular pH to adjust.[41]

Caution must be used when respiratory acidemia is permitted in patients with intracranial mass effects, recent myocardial infarctions, pulmonary hypertension, and, possibly, gastrointestinal bleeding.[41-43] Respiratory acidemia may also cause dyspnea and agitation in critically ill patients, requiring that they be heavily sedated or paralyzed.[41] In an animal model of acute lung injury, pulmonary shunt was higher when ventilation was achieved with smaller V_Ts.[44] Thus, low V_Ts may require an increased level of PEEP and/or F_{IO_2} to maintain acceptable levels of arterial oxygenation. As noted previously, in patients with ARDS who were matched to historical controls by physiologic scores, low-V_T strategy was associated with lower mortality.[28] Even more compelling is a recent preliminary report of a controlled trial using both low V_Ts and PEEP set above the lower P_{flex}. The results suggest trends in favor of this lung-protection strategy.[29]

In Conclusion

Considerable data support the existence and seriousness of stretch injury. Indeed, it is reasonable to assume that lung units 'designed' to withstand transalveolar pressures of 30-40 cm H_2O at maximum volume would be subject to injury, when distended beyond that level. Lung-protection ventilatory strategies using lower baseline pressure (PEEP) and smaller tidal excursions (V_T) require some concessions.

Lower baseline distention with PEEP may result in increased F_{IO_2} requirements and lower S_{aO_2} values and must be balanced against the requirement for PEEP high enough to maintain alveolar recruitment and reduce ventilator-induced lung injury. Similarly, lung protection offered by the use of strategies such as low tidal-volume ventilation and permissive hypercapnia must be balanced against the potential consequences of respiratory acidemia.

Both uncontrolled and preliminary reports from controlled trials using lung-protective strategies suggest their association with improved patient outcome. However, carefully conducted, randomized controlled studies are needed to fully assess the role of these approaches.

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MacIntyre Discussion

Hurst: Neil, I want to put you on the spot. You said from your slide that a pH of 7.15 is generally well tolerated. Okay, that's fine. Now, how low will you let it go? And if you let pH go below 7.15, what other variables should be measured at the bedside?

MacIntyre: I want to make sure I understood you, Jim. I haven't quite gathered the courage to go to 7.15. I've finally worked up the courage to go 7.2. Maybe in the next 6 months I'll get down to 7.15. We obviously don't do it in cerebral injury. I'm very reluctant to do it, as I said, in myocardial injury. Those are my two big concerns. So to ask me what I would do below 7.15—I'm not sure I know what I'd do because I haven't had the courage to go that low. I normally 'wimp out' and either deal with a larger tidal volume or some kind of a buffer or a frequency up to the point of air trapping. So I can't really answer your question because I've never treaded those waters, at least not intentionally.

Slutsky: We've treaded those waters a little bit. You're getting a little gutsier there, though, Neil. At the Consensus Conference,[1] I think you were willing to go down to 7.38. So, you're....

MacIntyre: At that Consensus Conference, Art did some fascinating things. One of them was an exercise with about 15 of us around the table, including Keith Hickling and John Downs. He just took a survey of what the comfortable 'minimum' pH was among these 15 people. I don't remember the exact numbers, but it was something like Hickling's 6.8 to John Downs's 7.35. You were kind enough to let me have the votes. I took them home and averaged them, and I think it was 7.30, if I remember correctly. Plus or minus 1.0. So, I went home and said this is the pH I'm going to be comfortable with because it was the average of what this so-called expert group decided.

Slutsky: Obviously, there are no correct answers. I've done some experiments with tracheal insufflation in apneic dogs. We used 100% O_2. We saw the P_{CO_2} rise, the pH drop, and I was sure (this was more than 10 years ago) that the animals would die at a pH of around 6.90. The pH kept dropping and dropping, and nothing happened until about 6.6, then the blood pressure started to tail off, and the animals died at pHs less than 6.5. Just think of this as a log plot! That's a *huge* increase in hydrogen ion concentration, so it made me a little less nervous about very low pHs. We've let the pH drop in some patients to about 7.0. At this point, I start to get a little nervous, although I'm not sure why.

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[1] Slutsky AE. Mechanical Ventilation. American College of Chest Physicians Consensus Conference, January 28-30, 1993. Chest 1993;104(6):1833-1859.

We don't use it on patients who may have increased intracranial pressure. We watch mainly for arrhythmias. Other than that, we don't do a lot. We don't give bicarb. When it gets a little lower than that I get nervous and increase the tidal volume a little bit, although I'm not sure that's the right thing to do. I think patients tolerate it, and, in a relatively short time, the pH starts to come up a little bit in most of them because of renal excretion.

MacIntyre: Intracellular buffering is actually pretty efficient. You can get a 'pretty whopping' acidosis in the blood, but inside the cells, there are a number of mechanisms that actually allow fairly rapid compensation for pH shifts.

Hurst: Suppose you have a patient with a pH of 7.0 and adequate systemic oxygen delivery. The arterial-venous oxygen difference has not widened from the previous levels. Why wouldn't you be comfortable at that level? Or would you?

MacIntyre: Well, maybe I should be comfortable, Jim. I must say if I live by my rule of 7.2, I would be forced to either wimp out on tidal volume or go look for a buffer, either bicarb or THAM or something. But maybe you're exactly right. Maybe I am being overly cautious, and, as you say, if blood pressure and oxygen delivery and cardiovascular status are 'cool,' maybe I should just leave them alone and continue to try to protect the lung from high-stretch injury. Certainly Keith Hickling would say that.

Hurst: One other comment about plateau pressures. As Rich (Branson) will tell you, we have for a long time been interested in intra-abdominal pressure and its relationship to ventilatory and renal compromise. The thing that bothers me about an absolute plateau pressure of 35 is that in patients with what has come to be known in surgical circles as abdominal compartment syndrome, I think all bets are off. One of two things is going to happen. Either we measure intra-abdominal pressure very carefully, very religiously, and decompress the abdomen when intra-abdominal pressure reaches 25 mm Hg, or we live with a plateau pressure that is somewhat higher. And what's somewhat higher? I don't know exactly what that upper plateau is. As long as the patient is reasonably well ventilated and urine output is reasonable, we tend to let that plateau drift up a little bit. Wouldn't you say, Rich?

Branson: Yes, Jim. I have a couple of questions for Neil. I'll get back to the PEEP issue too. Permissive hypercapnia is a technique that I'll have to admit we talk about doing in our ICU, but we never have anybody who has a problem eliminating CO_2. Our patients are mostly healthy, young trauma patients and some elderly patients after emergency surgery. Suppose you have someone with severe ARDS who has had a pH of 7.25 or maybe 7.3 for several weeks, and a P_{aCO_2} of 80. What is the effect of that high CO_2 on weaning, and is there any effect on weaning time because of that excessive CO_2?

MacIntyre: There probably is a theoretical effect because the pH has been readjusted by renal mechanisms, but I don't think anybody has ever looked at what it's going to do. I agree with you that, theoretically, that may be an issue.

Branson: It would seem to me that it might actually prolong weaning. Bob (Kacmarek) or Dean (Hess), somebody who does this—I don't know because we don't do it?

Slutsky: Let me talk theoretically, first. I'm not sure that it necessarily has to prolong weaning. I could make an argument (I'm not sure I believe this argument) that it might actually quicken the weaning process, if you're 'gutsy' enough. Suppose the P_{CO_2} is 100 mm Hg, the patient is stable, and the pH approaches normal values. The minute ventilation that person needs now to keep the P_{CO_2} at 100 mm Hg is about 40% of what would be needed to keep it at 40. So that might be a type of patient you could take off the ventilator. They could be fine on their own, with supplemental oxygen because their ventilatory requirements are down substantially. Then they could take care of the rest themselves. We don't do that. We don't usually extubate people with P_{CO_2}s of 100—don't get me wrong. But it seems that when a patient gets better the P_{CO_2} and pH seem to quickly get taken care of. It's hard to answer specifically. Does it lengthen the weaning? These patients, by definition, are the sickest patients; so, yes, if you look at it anecdotally their weaning may be longer, but that may be because they're sicker.

MacIntyre: I think that is a fair point. At least in our hands by the time the patient's ready for weaning, the P_{CO_2} has come back to a more reasonable range.

Kacmarek: I think that's the key. As time goes by, CO_2 comes back down if you pay attention to the ventilatory strategy. Although we've had patients in whom this was a problem, most of the time they were patients with underlying chronic lung disease who have acclimated to the high CO_2 and have had a difficult time when we wanted to wean them. P_{aCO_2} did not readjust as rapidly as expected. But in my experience, in most patients just as P_{aCO_2} goes up gradually, it comes down gradually to baseline as the patient improves.

Hess: You don't usually start to wean them when their P_{CO_2} is 90 torr and their pH is 7.2. By the time you start to wean them, you've usually fixed a lot of other things; so, they aren't at their peak level of permissive hypercapnia. If I could get back to the thing that Jim (Hurst) brought up and Art (Slutsky) and Rich (Branson) remarked on, regarding the high intra-abdominal pressures and whether you can allow the plateau pressures to go up. The thing that's bothering me about that is—and maybe some of you can help me with this—if your abdominal pressure goes up, from ascites or whatever, how is that pressure transmitted to the pleural space? Is the pressure transmitted equally throughout the pleural space or do you end up increasing the pleural pressure in the bases so you increase the plateau pressure and just overdistend the more apical parts of the lung? Is my question making any sense?

Hurst: Yes, it's making a lot of sense.

Hess: I've wrestled with that, and the question has come up in our ICU. This guy's abdomen is distended, so can we accept higher plateau pressures? My response has been I don't know because I don't know how that pressure is going to be transmitted to the pleural space.

Hurst: I don't know of anybody who knows, do you Rich (Branson)? One can assume that these patients are nursed in the supine position. We're not talking about patients with ascites. What we typically see is the patient with a huge retroperitoneal hematoma or a postresuscitative diffuse edema. Any attempt to close these patients' abdomens results in a huge increase in intra-abdominal pressure. When you look at the chest x-ray, the diaphragm is not lower than maybe the 3rd or 4th intercostal space. One would presume that at that point pulmonary parenchymal compliance is reasonably normal. The reason I say that is that if you take them back to the operating room and decompress the abdomen, all of a sudden the peak inflation pressures come down, plateau pressures come down. If you were ventilating them at one plateau, then at that same plateau pressure, resulting tidal volume is much larger. I'm not sure that we can say the same thing for the patient who has ARDS and who then has an increase in intra-abdominal pressure. I don't know that anybody can.

Peruzzi: We've recently begun a liver transplant program, and we've been dealing with patients like this. In terms of the transmission, I think that there are two aspects—one is the diaphragmatic pressure transmitted from the abdomen and the other is the accumulation of pleural effusion that comes with intractable ascites. Both of those things are extrinsic mechanical restrictive processes that tend to be more uniformly applied. When this situation develops, the abdominal and pleural fluid tends, I think, to create a more global increase in intrathoracic pressure. In that circumstance, higher pressures can be tolerated because alveolar overdistention is not the primary concern with the increased pressures.

Slutsky: I think the pleural pressures do go up, Dean, and that it is reasonably safe to use substantially higher pressures. The ideal approach is to use an esophageal balloon to provide an estimate of pleural pressure—you can then go quite a bit higher on airway pressures, monitoring transpulmonary pressure. In fact, as Jim said, if you have lungs that look like they're well below their FRC, even though the inflation pressures are high, the outside pressure, the pleural pressure, is substantially higher.

Adams: We've been talking about regions of injury. I have a question/comment about injury usually occurring in the dependent segments. How is it that placing a patient prone seems to lead—in at least half the cases—to an improvement? Do you believe it's due to redistribution of blood flow because of the prone position or because of the decrease in the range of pleural pressures from the top to the bottom that is seen when you place patients prone?

MacIntyre: Wow! Placing a patient prone is a fascinating concept. It's terrifying to the nurses in our ICU that we would even consider such a maneuver. But I think Gattinoni has clearly shown that things do change dramatically when you go into the prone position.[1] Clearly, blood flow now goes toward the sternal part of the chest, and I think the hydrostatic edema effects are also shifted to those newly dependent regions of the lung. In fact, maybe the best way to handle somebody would be to put them on a rotisserie, so that they are constantly turning—over a 24-hour period of time or something of that nature. I'm not sure it's anything more complicated, though, than just shifting of edema fluid and blood flow from one region of the lung to another. Maybe somebody else feels differently. Jim?

1. Gattinoni L, D'Andrea L, Pelosi P, Vitale G, Pesenti A, Fumagalli R. Regional effects and mechanism of peak end-expiratory pressure in early adult respiratory distress syndrome. JAMA 1993;269(16):2122-2127. *See the published erratum JAMA 1993;270(15):1814.*

Hurst: No, I was just going to say that when I was in San Diego, Dick Peters and Dick Virgilio were very interested in just this issue. There was a time when we had patients with ARDS whom we either nursed on a circle electric bed (for those of us who are old enough to remember those) or on Stryker frames. While you did realize some transient improvement, that improvement was not sustained. I guess the thing that made us the most nervous, in addition to the nurses being nervous, was that when you lost a critical airway in patients, face down, it is a real disaster. The combination of lack of sustained improvement and the airway management issues led us to abandon that technique. Of course, our understanding in the late 60s was a lot different than it is now. So maybe it's worth revisiting.

Slutsky: Rick Albert and his group have published a number of things[1-3] on prone position, and some studies in dogs. It seems that the prone position is better. However, dogs are different, you know. They have a funny shaped chest, and I think the gravitational factors plus the geometry don't make that a great model for humans. Whatever the inflammatory response or whatever the damage when you're nursing someone supine, when you then turn them prone, they're going to be better for a little while, but my guess is that they are going to get worse again. Our concerns are exactly the same in terms of the nursing. I think those are real concerns.

1. Albert RK, Leasa D, Sanderson M, Robertson HT, Hlastala MP. The prone position improves arterial oxygenation and reduces shunt in oleic-acid-induced acute lung injury. Am Rev Respir Dis 1987;135(3):628-633.
2. Wiener CM, Kirk W, Albert RK. Prone position reverses gravitational distribution of perfusion in dog lungs with oleic acid-induced injury. J Appl Physiol 1990;68(4):1386-1392.
3. Glenny RW, Lamm WJE, Albert RK, Robertson HT. Gravity is a minor determinant of pulmonary blood flow distribution. J Appl Physiol 1991;71(2):620-629.

Branson: Neil, on another subject—Is the increase in PEEP—when you have the same plateau pressure—protective because you don't have the opening and closing? Is PEEP protective of the lung injury? Or, is it simply the fact that when you go from zero to 10 cm H_2O of PEEP and your peak pressure is still 45 cm H_2O that you're pressure swing now is only 35?

MacIntyre: The question is clear, the answer is not. If you put PEEP in there and the plateau stays exactly the same, that implies you've really improved compliance and probably recruited a good hunk of lung. In so doing, you have not increased the maximal stretch at all because the plateaus remain the same, and you probably have induced some lung protection from the opening and closing. One could even argue that the tidal distention or the tidal pressure swings in the healthier alveoli that weren't collapsed are going to be less now, too. That may be also protective. But a lot of this is speculation.

Slutsky: There's a paper by Corbridge[1] and his group in which they addressed that—in a way. They increased the PEEP and found decreased evidence of lung injury. Their discussion revolved around the concept that you're talking about. The pressure swings were smaller, so the $\Delta V/\Delta P$ was a lot less. I think, however, that the opening and closing (rather than the swings of pressure) is what is important although I don't know of any studies that specifically have addressed that by increasing the peak pressures the equivalent amount.

1. Corbridge TC, Wood LD, Crawford GP, Chudoba MJ, Yanos J, Sznajder JI. Adverse effects of large V_T and low PEEP in canine acid aspiration. Am Rev Respir Dis 1990;142(2)311-315.

MacIntyre: From Dreyfuss's data,[1] if you glue a couple of his figures together, you can see that for the same peak pressures of 45 cm H_2O, if you generate that peak pressure with a small tidal volume and a lot of PEEP, as opposed to *all* tidal volume and no PEEP, you get much less injury with the small tidal volume/PEEP than with the large tidal volume-no PEEP.

1. Dreyfuss D, Saumon K. The role of tidal volume, FRC, and end-expiratory volume in the development of pulmonary edema following mechanical ventilation. Am J Respir Crit Care Med 1993;148(5):1184-1203.

Peruzzi: I think that is borne out in that last study that I talked about—Gattinoni[1] looking at the distribution of tidal volume. He looked at alveolar recruitment in association with tidal ventilation, plateau pressure, and, also, with PEEP and found that indeed, at least by CT scans, that the amount of recruitment going on in the middle lung segments was decreased with PEEP applied. The upper lung segments were not overdistended any further, and the lower lung segments still had a reasonable amount of recruitment going on, as much as was going on with the tidal volume. But by and large, the application of PEEP did obliterate that collapse and opening associated with the tidal ventilation.

1. Gattinoni L, D'Andrea L, Pelosi P, Vitale G, Pesenti A, Fumagalli R. Regional effects and mechanism of peak end-expiratory pressure in early adult respiratory distress syndrome. JAMA 1993;269(16):2122-2127. *See the published erratum JAMA 1993;270(15):1814.*

Slutsky: That's the easy answer to your problem. Sort of tongue-in-cheek, I say just do CT scans on everybody at different lung volumes at multiple time points.

Kacmarek: If you go back to the Webb and Tierney article from 1974,[1] the group they ventilated at 45 cm H_2O with no PEEP had the most injury. In the 2 other groups, both with 10 cm H_2O PEEP but one with 30 cm H_2O and one with 45 cm H_2O as a peak pressure, the injury was pretty similar and much less than that in the group without PEEP. This is the only study that I know of that addressed the issue of looking at PEEP and the difference in total stretch on the extent of injury.

1. Webb HH, Tierney DE. Experimental pulmonary edema due to intermittent positive pressure ventilation with high inflation pressures: protection by positive end-expiratory pressure. Am Rev Respir Dis 1974;110(5):556-565.

MacIntyre: I think what this is showing us is that all of these effects are probably playing a role—collapse, stretch, frequency, acceleration, duration—all of them are probably playing a role and, depending upon study design, one is going to have a predominance over the other.

Decision Support Systems for Management of Mechanical Ventilation

Thomas D East PhD and Alan H Morris MD

Introduction
Tools for Decision Support
  Education & Training
  Reference & Training Materials
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Introduction

Management of present-day mechanical ventilation can be a complex, sophisticated process. We recently went to the bedside of a critically ill patient on mechanical ventilation and counted the number of variables that were reviewed for clinical decision making (eg, F_{IO_2}, V_T, ventilator rate, peak pressure, and others). These were not repeated measurements—systolic blood pressure only counted once even though it was measured several times. The total was in excess of 236 variables![1] Table 1 summarizes the number of options for settings on mechanical ventilators. This total includes only 7 common settings. It does not include the more esoteric settings available on many mechanical ventilators today. Despite this simplification, more than 37 billion permutations are available from these 7 mechanical ventilator settings. In a real clinical situation, one would not have as many different types of ventilators or modes to choose from and would normally only change the settings in discrete steps (eg, F_{IO_2} in 10% increments). This reduces the number of possibilities to 1.3 million. When a clinician makes a decision about changing ventilator settings, it may only be necessary to choose whether to increase or decrease the setting or to make no change at all. This dramatically simplifies the problem, leaving only 972 permutations. If one argues that all that needs to be decided is whether to make any change, only 128 possible combinations remain. Even if one considers all of the settings to be independent of one another (eg, the effect of positive end-expiratory pressure [PEEP] =15 cm H_2O is the same, regardless of ventilator, mode, or F_{IO_2}), the smallest number of choices is 14.

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A version of this paper was presented by Dr East during the RESPIRATORY CARE Journal Conference, Mechanical Ventilation: Ventilatory Techniques, Pharmacology, and Patient Management Strategies, held in Cancún, Mexico, October 6-8, 1995.

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The research on decision-support systems for mechanical ventilation was supported by The Agency for Health Care Policy and Research grant HS06594, LDS Hospital, Intermountain Health Care, the Deseret Foundation, Siemens Life Support Systems, Emtek (A Division of Motorola), and Microhealth Systems Inc. Research on closed-loop control was supported by Hamilton Ventilators, Bonaduz, Switzerland.

Table 1. The Complexity of Decision Making in Mechanical Ventilation

Variable	Global Possibilities*	Clinical Scenario	↑, ↓, or No Change	Change? Y/N
Ventilator	15	3	2	2
Mode	10	5	2	2
F_{IO_2}	79	9	3	2
Tidal Volume	50	10	3	2
Ventilatory Rate	45	12	3	2
Peak Flow or I:E	35	8	3	2
PEEP	40	10	3	2
Permutations	37,327,500,000	1,296,000	972	128
Sum	274	57	19	14

*Global possibilities include all options for changes (eg. 79 possible choices of F_{IO_2},0.21,0.22,0.23. 0.98,0.99,1.00), whereas the Clinical Scenarios options are the number that would be clinically feasible (F_{IO_2} is only changed in 10% increments). The column with the up and down arrows show the number of decisions possible when a variable can only increase ↑, decrease ↓, or remain unchanged. The last column has only 2 options for each setting. The Permutations totals assume that the variables interact whereas the Sum totals assume that the variables do not interact

Psychologists have known for years that human beings have a limited capacity for processing parallel information. They have created a rule that is known as the "magic number 7 ± 2."[2] This implies that humans can only process 7 simultaneous streams of information. How is it possible to manage a ventilator on a critically ill patient with more than 200 variables and a thousand or more possible choices for settings? David Eddy summarized it

> It is simply unrealistic to think that individuals can synthesize in their head scores of pieces of evidence, accurately estimate the outcomes of different options, and accurately judge the desirability of those outcomes for patients.... All confirm what would be expected from common sense: The complexity of modern medicine exceeds the inherent limitations of the unaided human mind.[3]

Decision support tools for management of patients on mechanical ventilation are clearly desirable.

Tools For Decision Support

Figure 1 illustrates the categories of decision support available for mechanical ventilation: education and training, reference materials, conceptual frameworks, cookbooks or rules of thumb, guidelines, care paths, clinical protocols and specific executable protocols. Figure 1 also describes the distribution of resources among these different tools. Clearly, education and reference materials are the primary foundation; however, in recent years guideline development has grown into a huge effort throughout the U.S. Interest is growing in the development and use of clinical protocols. The most basic endeavor is the creation of specific executable protocols. However, the ability of these tools to standardize care is inversely related to their position on the pyramid—education and training provide the least amount of standardization and specific executable protocols provide the highest level of standardization.

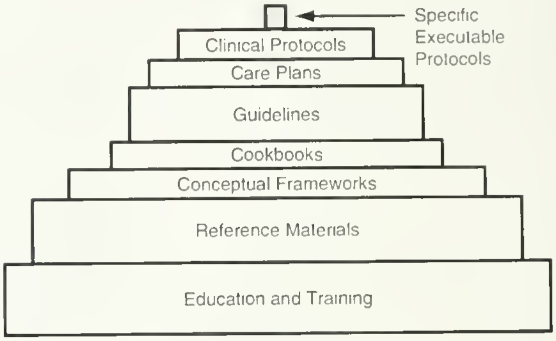

Fig. 1. Decision support tools available for mechanical ventilation. The area of each rectangle is meant to imply the relative distribution of resources allocated to develop these different tools.

Education & Training

Education and clinical training are at the heart of helping clinicians manage patients on mechanical ventilation. This category includes college, graduate school, medical school, local, regional and national meetings, and other continuing education programs. This decision-support tool lays a solid foundation; however, it is not a tool that can easily referred to in a clinical setting, unless one has a good memory. Traditional education does not provide any level of standardization in the process of management of mechanical ventilation. It may actually promote the opposite because each training program, and even each professor in the program, may impart a unique perspective to students.

Reference & Training Materials

Reference materials are tools that provide a link between education and training and clinical practice. One can find books, journals, videotapes, and other forms of reference materials in medical libraries and on individuals' bookshelves. These tools are typically not available at the point of care and are infrequently referenced during the decision making process at the bedside. Some pocket reference guides are attempts to bridge this gap. These tools do not provide any level of standardization, and they cannot provide quick and easy help for solving a unique clinical problem (eg. at 2 AM, Mrs Jones's P_{aO_2} is only 45 torr, and PEEP is already 15 cm H_2O with F_{IO_2} 1.0. What do you do?).

As computers become more commonplace at the point of care, access to electronic reference materials becomes more feasible. A variety of World Wide Web pages and list-server groups are evolving in the field of respiratory care and contain information on management of mechanical ventilation. The National Library of Medicine [4] as well as several other integrated academic information management system (IAIMS) research sites have been working hard to integrate reference databases such as MEDLINE, and even full text references, into the everyday work environment of the hospital. It has been suggested that eventually information systems would issue context-sensitive searches automatically. For example, when the clinician examines data from the mechanical ventilator, the computer might issue a query for current references explaining the interpretation of the data and therapeutic options. If the clinician needs help at any point, he or she might only need to push a button to receive both text and graphic information. Training and teaching processes at the bedside could be integrated with patient care. For example, if the clinician were reviewing a pressure-volume loop on the computer and wants more information on where the inflection points are and what they mean, the computer could retrieve the latest journal articles and enter into a multimedia training program that would describe, at the user's discretion, many different levels of pulmonary function measurement and interpretation. The multimedia program might include color images of devices and anatomy, typical auscultation sounds, graphs, and text. This type of computer-based learning is not new.[5] The advantages of computer-based instruction as a method of independent learning were highlighted in the recommendations of the Association of American Medical Colleges' 1984 report of the Project Panel on the General Professional Education of the Physician and College Preparation for medicine (GREP).[6,7] In fact, systems have already been designed for teaching pulmonary auscultation,[7] interpretation of blood-gas and acid-base data,[8] diagnosis of chest pain,[9] diagnosis of acute respiratory failure,[10] and airway management,[11] along with a general model of the pulmonary system known as MacPuf.[12] Although the results of these studies do not conclusively demonstrate that computer-based learning is better than traditional training, this field is still in its infancy, and it is commonly believed that learning at the point of care will be far more valuable than traditional lecture and book formats.[6,7]

Conceptual Frameworks

Conceptual frameworks are descriptions of ventilator-management strategies. These are typically found in review articles or books that summarize the topic. They are rarely specific and use only the broadest of strokes in defining how to actually manage patients on mechanical ventilation. The following is an example from the latest edition of the Handbook of Critical Care.[13]

> …provide sufficient oxygen to the tissues of the body … to increase the P_{aO_2} to 60-70 torr … ARDS [acute respiratory distress syndrome] may also require the addition of PEEP.

Whereas these tools may provide a general conceptual framework for what must be done, they do not tell the clinician *how* to provide sufficient oxygen to the body. What mode, F_{IO_2}, and PEEP should be used? What should be assessed and when? Typically these reviews, even comprehensive ones, are not used at the point of care and provide only marginal assistance for medical decision making.

Cookbooks, or Rules of Thumb

Cookbook rules (or rules of thumb) are used in medicine. They are also criticized as not being focused on the individual patient. Some authors have essentially equated cookbook rules with poor quality medicine. Management of mechanical ventilation is filled with these cookbook approaches. A good example is the 12/12 rule: $V_T = 12$ mL/kg body weight at a ventilator rate of 12 breaths/min.[14] A prevalent rule throughout the respiratory care community is that plateau pressures higher than 35 cm H_2O are harmful and should be avoided at all costs.[15] These rules are appealing because they are easy to remember and to bring to the bedside as aids for specific patient-management decisions. However, they do not provide much standardization because they are not consistently followed.

Clinical Practice Guidelines

A huge effort nationwide is focused on the generation of guidelines for many different areas of medicine. Various organizations and government bodies have constructed more than 4,000 clinical guidelines.[16] The Agency for Health Care Policy and Research has had several large programs focused on developing and disseminating clinical practice guidelines.[17] The American Association for Respiratory Care (AARC) has also produced a large set of clinical practice guidelines focused on respiratory care. In particular, they have produced the following guidelines related to mechanical ventilation:

- Endotracheal Suctioning of Mechanically Ventilated Adults and Children with Artificial Airways[18]
- Humidification during Mechanical Ventilation[19]
- Patient-Ventilator System Checks[20]
- Management of Airway Emergencies[21]
- Ventilator Circuit Changes[22]
- Metabolic Measurement using Indirect Calorimetry during Mechanical Ventilation[23]
- Neonatal Time-Triggered, Pressure-Limited, Time-Cycled Mechanical Ventilation[24]

Each of these represents an immense effort to review the current state of the art and prepare a guideline. For example, the neonatal time-triggered, pressure-limited, time-cycled mechanical ventilation[24] guideline contains 118 numbered sections, cites 117 references, and is 9 journal pages long. Clearly, this is an important tool and does provide useful information to support decision making. However, guidelines are typically filled with "weasel words"[25] that make them ambiguous and not likely to standardize care or provide solid decision support. Following are some examples from the AARC guidelines. The weasel words are in type that is both boldface and italic.

> **Blood-Gas Assessment:**
> 11.2.1 ***Periodic*** sampling of blood-gas values by ***arterial, capillary or venous*** route. P_{aO_2} ***should be kept below 80 torr***....[24]
> **Ambiguities:** How often is periodic, 2 hours, 4 hours, or what? How can one ensure that P_{aO_2} is < 80 torr if only capillary or venous blood is drawn? How far below 80 torr should P_{aO_2} be maintained? What is too low?
>
> **PEEP Adjustment:**
> 11.5.4 PEEP increases FRC [functional residual capacity] and ***may improve*** oxygenation and ventilation-perfusion relationship. (PEEP is ***typically*** adjusted at 4-7 cm H_2O—levels beyond this range ***may result in hyperinflation***.)[24]
> **Ambiguities:** What PEEP level should be used—4-7 cm H_2O? If so, what PEEP level between 4 and 7 cm H_2O should be used? If the patient is still hypoxemic, should PEEP be increased beyond 7 cm H_2O? Is there another way to deal with hyperinflation (eg, lengthen expiratory time)?

Decision-support tools such as these are more specific than conceptual frameworks. However, they do not provide specific guidance that can be put into use at the point of care. The AARC has stated clearly that these Guidelines were never intended to provide decision support at the point of care.[26] They do, however, provide a good foundation for further decision-support-tool development—eg, patient-driven protocols and clinical pathways.

Evidence supporting the use of guidelines is scarce. This lack of evidence has sparked concern that the immense investment in guidelines may be ineffective in truly improving the quality of care and reducing costs.[16] An Office of Technology report on the status of federal programs for guideline development[17] concluded "...the federal effort has been largely unsuccessful at its initial charge: evaluating the comparative effectiveness of alternative treatments." The Office of Technology Assessment found that guidelines often overlap and conflict.[17] This is true both within different government agencies as well as in the private sector. The American College of Physicians recently conducted a survey regarding guidelines.[16] Of the respondents, 70% believed that guidelines would improve the quality of care; however, their familiarity with various common guidelines ranged from 11% to 59%. The authors conclude that "guidelines often do not affect clinical practice or health outcomes." Many believe that the issue is implementation—how do you convert guidelines to protocols and integrate them into everyday practice at the point of care?[16]

Care Paths

Care paths are popular with nurses and with the 'gatekeepers' of health insurers. An example of a care path related to mechanical ventilation is

> Post thoracic surgery patients should be weaned and extubated within 24 hours.

In general, care paths are based on observing the performance of a representative sample. This particular example was derived by looking at time to extubation in a large group of patients. The time at which the majority of patients in the group were extubated suggested 24 hours as the 'milestone' in the care path. Care paths can help the clinician at the point of care by outlining a general course. However, they do little to help with decisions that must be made on a moment-to-moment basis for individual patients. For example, what should be done about Mr Doe who underwent coronary artery bypass surgery 23 hours ago? He is now experiencing anxiety, is having difficulty clearing his secretions, and, overall, is struggling with the weaning process. Should he be extubated in an hour? Will he be able to protect his airway? Care paths are not likely to provide the answer. Care paths can be easily integrated directly into the patient's bedside chart to be available at the point of care. New approaches to care paths incorporate adjustments based on the patient's illness severity to tailor them to the individual.[27]

Clinical Protocols

Clinical protocols are sometimes referred to as therapist-driven or patient-driven protocols. These protocols tend to be more specific than guidelines; however, they do not provide completely unambiguous instructions. The AARC maintains a protocol clearinghouse, and, as of September 15, 1995, at least 8 institutions had submitted protocols for managing patients on mechanical ventilation. Among these, protocols for uncomplicated weaning are probably the largest subset.[28-30]

Some evidence attests that such protocols improve the quality of care and reduce costs in other areas of respiratory care.[31] Only a small number of studies on the efficacy of clinical protocols for mechanical ventilation have been completed. Wood et al[30] demonstrated that weaning protocols were slightly more effective than their historical controls. Despite the lack of evidence of efficacy, the use of protocols is increasing throughout the respiratory care community.[32] The large number of protocols on the same subjects in the AARC clearinghouse indicate that the problems of duplication and contradiction exist.[16,17]

Specific Executable Protocols

Specific executable protocols contain knowledge-based instructions that are complete, clear, and concise—they are not ambiguous. The knowledge can be represented in a variety of different ways. The knowledge may be represented as a set of rules such as "If A > 2 then do B," or it may be expressed as a probability: "If A > 2 then the probability of B is 0.6." The concept of specific executable protocols is not new—they have been available for some medical applications for more than 20 years. In general, specific executable protocols can be separated into two categories, diagnostic and therapeutic.

Diagnostic specific executable protocols have been developed for the interpretation of blood-gas data.[33-38] Hingston et al[36] performed an interesting study illustrating that although 61% of the participants at medical grand rounds believed that they knew how to interpret blood-gas data, and 71% believed that an expert system was unnecessary, when tested they were able to answer only 39% of the questions correctly. These results indicate that clinicians are not capable of remembering everything in infinite detail and that bedside decision support may be a important asset. Other diagnostic systems include a system for community-acquired pneumonia,[39] occupational lung disease,[40] interstitial lung diseases,[41] and breath detection from flow and capnography data.[42]

Several different specific executable protocols have been developed for ventilator management.[43-55] These specific executable protocols can be divided into those designed for management of the mechanical ventilator in general[43-51,55] and those that are designed specifically for weaning.[52-54]

At the LDS Hospital, the development of specific executable protocols for management of mechanical ventilation was originally stimulated by investigator needs during a recent $ECCO_2R$ (extracorporeal CO_2 removal) clinical trial.[56] Our intent was to develop protocols to standardize therapy.[1,57] We developed a special protocol for management of ventilation and intrinsic PEEP during pressure-controlled inverse-ratio ventilation.[57] New protocols have been written for ventilation and pH control.[58]

The protocols were developed using an iterative approach that involved the clinical environment as an integral part of the development process. The protocols were computerized using the HELP system at the LDS Hospital.[1,49-51,57,59] As shown in Figure 2, the computer displays instructions on the terminal at the bedside. A member of the clinical care team, typically the respiratory therapist, reads the instructions and makes the indicated ventilator-setting adjustment. This is not a closed-loop system. The computer never makes any adjustments in therapy directly. The clinical care team members always have the option to refuse an instruction. However, they must provide a reason for not following an instruction. This provides feedback that is critical for protocol refinement.

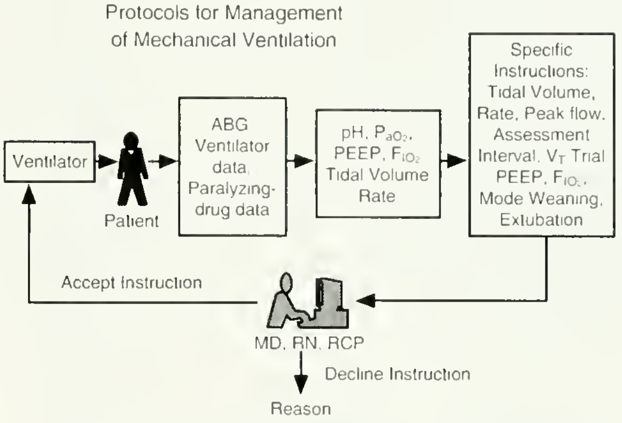
Protocols for Management
of Mechanical Ventilation
Ventilator
Patient
ABG Ventilator data, Paralyzing-drug data
pH, P_{aO_2}, PEEP, F_{IO_2} Tidal Volume Rate
Specific Instructions: Tidal Volume, Rate, Peak flow, Assessment Interval, V_T Trial PEEP, F_{IO_2}, Mode Weaning, Extubation
Accept Instruction
MD, RN, RCP
Decline Instruction
Reason

Fig 2. Diagram illustrating the function of the computerized clinical protocols for mechanical ventilation developed at LDS Hospital. Arterial blood-gas values and respiratory care data flow into the computer via routine computerized charting. The protocols run and produce a set of instructions that are stored in the patient's database. These instructions include mode, tidal volume (V_T), ventilator rate, F_{IO_2} and positive end-expiratory pressure (PEEP), I:E, peak flow, weaning steps and extubation, and the timing and nature of required patient assessments. The clinician then reviews the instructions, decides to accept or decline the instructions, enters a defense for declined instructions on the computer at the patient's bedside, and adjusts the ventilator, beginning a new cycle.[49]

We evaluated the ability of the protocols to control care during around-the-clock application in the intensive care unit.[60] This was the equivalent of a Phase-2 clinical trial. The study was approved by the LDS Hospital Research and Human Rights Committee. Patients with ARDS (n = 111) were enrolled in the study. Outcomes were compared to those of three historical controls: patients from the LDS Hospital prior to the use of protocols, from the Massachusetts General Hospital (MGH) between 1978 and 1988,[61] and from the European Collaborative Study.[62]

Figure 3 summarizes the results of using the continuous positive-pressure ventilation oxygenation protocol in 111 ARDS patients.[49,60-63] Fewer than 1% of the instructions generated were disregarded due to a challenge of the medical logic. The survival in this group was higher than that previously seen in any of the control groups. The survival of the subsets of the 111 patients—those who met or did not meet entry criteria for extracorporeal membrane oxygenation—was also higher than those published previously.[61] We conclude that these protocols can be used effectively and safely to standardize the management of mechanical ventilation. The protocols were associated with an increase in survival and a randomized, controlled, prospective, multicenter clinical trial is underway to objectively compare this protocol with physician-directed care

(AHCPR grant HS06594). This study seeks to answer two questions: (1) Can a decision support system be exported to other centers and used by clinicians who have not been involved in its development? and (2) Does the use of such a system affect patient outcome? Patients with ARDS ($n = 400$) will be enrolled. We intend to evaluate the efficacy of this system using a hierarchical 4-level approach: survival, length of ICU stay, morbidity, and the incidence and severity of barotrauma. We will determine the generalizablity of the computerized decision support system by examining the percentage of time in the trial during which protocols controlled patient care, the number of protocol instructions that were not followed, and the number of objections to protocol logic, which, based on medical evidence, forced a change in the logic.

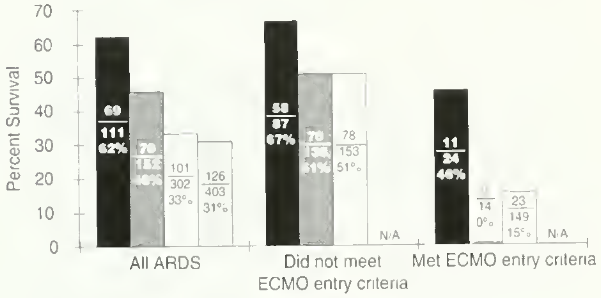

Fig. 3. Survival results of the continuous positive-pressure ventilation oxygenation protocol use in 111 ARDS patients in the shock-trauma intensive care unit at LDS Hospital 1987-1992[63] ■ and 1983-1985[60] ▨, Massachusetts General Hospital 1978-1988[61] □, and the European Collaborative Trial □.[62]

We believe that these computerized protocols can be used to successfully manage mechanical ventilation in critically ill patients. To our knowledge, this is the first demonstration that a computerized decision support system can be used effectively in the critical care environment. We envision that this system could be used in several different settings: (1) to control future clinical trials as part of the ongoing, nationwide effort to determine the efficacy of existing and new therapies; (2) to standardize the management of patients on mechanical ventilation—an essential part of the continuous quality improvement process; and (3) to provide decision support in the management of mechanical ventilation for patients with ARDS who are in a variety of settings where no expert in management of patients with ARDS is available.

Closed-Loop Control

Closed-loop control is similar to a specific executable protocol except that the computer directly adjusts the mechanical ventilator. A closed-loop control system for mechanical ventilation was first introduced in 1953 by Frumin et al.[64] This system used electronic control systems but not a computer. This sophisticated system included oxygen and carbon dioxide analyzers and adjusted the F_{IO_2} and V_T during anesthesia. Several other analog closed-loop control systems were reported between 1953 and 1973.[65] Coles et al[66] introduced the first computerized closed-loop controller for mechanical ventilation. This system controlled F_{IO_2} and tidal volume during anesthesia. Several other systems were designed within the last 20 years for closed-loop control of V_T and ventilatory rate based on expired CO_2 monitoring.[65,67-70] Most systems were designed to maintain a desired end-tidal CO_2 value. Although all of these systems worked well, they have not been used much clinically because of the differences between end-tidal CO_2 and P_{aCO_2} in patients who have large physiologic dead space.

Some closed-loop controllers have been designed for managing oxygenation. Most of these systems are simple systems designed to adjust F_{IO_2} to produce a desired P_{aO_2} or oxyhemoglobin saturation by pulse oximetry (S_{pO_2}).[71-73] Morozoff et al[71] demonstrated that, in neonates, computer control was able to maintain a desired S_{pO_2} with more accuracy than manual control. My colleagues and I[74] designed a system for control of PEEP based upon normalizing FRC. Strickland and Hasson developed a closed-loop controller for weaning patients on synchronized intermittent mandatory ventilation (SIMV) with pressure support (PS) in postoperative cardiac patients.[54,75] This controller decreased the SIMV rate by 2 breaths/min every 5 minutes until a rate of 2 breaths/min was reached, then decreased pressure support by 4 cm H_2O every 5 minutes as long as the patient met the following criteria: respiratory rate 8-25 breaths/min, $\dot{V}_E$ 6-14 L/min, and $S_{pO_2} \geq 90\%$. This rule-based controller automatically adjusted a Puritan Bennett 7200 ventilator (Puritan Bennett Corp, Overland Park KS). This system was used in a 15-patient prospective, randomized trial (9 had computer control, and 6 had manual control). The results showed that the computer-controlled group had significantly fewer blood gases (mean ± SD, 1.4 ± 0.7 vs 7.2 ± 4.3, $p < 0.05$), had shorter weaning times (mean ± SD, 18.7 ± 5.9 vs 25.6 ± 5.6 h, $p < 0.05$), and experienced less time outside acceptable ventilator-rate and V_T bounds (mean ± SD, 3.2 ± 2.8 vs 6.6 ± 4.1 min, $p < 0.05$).[75]

Despite several clever and sophisticated systems, none of these closed-loop controllers for mechanical ventilation have had a major impact on clinical care. There are two primary reasons for this. The first is that these systems are relying on input data from sensors (eg, P_{aO_2}, end-tidal CO_2, transcutaneous P_{O_2}, S_{aO_2}) that are too unreliable to trust in a closed-loop system; Second, most of these systems were designed by engineers in the laboratory, and, whereas they are excellent engineering exercises, they are not closely linked to common clinical practice.

Recently, we tried to address this problem by designing a system, based on well-established protocols for management of mechanical ventilation,[50,51,57] that provides continuous closed-loop control of oxygenation[76] using a Hamilton

Amadeus ventilator (Hamilton Medical Inc, Reno NV) controlled by an Apple Macintosh SE-30 computer (Apple Computer Inc, Cupertino CA). A Shiley Continucath (Shiley Inc, Irvine CA) intra-arterial electrode was used to provide continuous measurement of P_{aO_2}. Arterial oxygen saturation (S_{aO_2}) was calculated from the P_{aO_2}, pH, P_{aCO_2}, and temperature using standard equations. The magnitude and direction of a therapy change were determined by the P_{aO_2} error (difference between measured and desired P_{aO_2}). The overall gain of the controller was a function of the S_{aO_2}. This control strategy provided an aggressive response to hypoxemia and a more conservative response to hyperoxia. The content of the therapy change was determined from a fifth dimensional quality surface that described the tradeoff between PEEP and F_{IO_2} changes for a specific combination of current PEEP, F_{IO_2}, and P_{aO_2} values. The quality surface was generated from clinically proven protocols. The controller was tested in an animal model of ARDS and was shown to be safe and effective.[76] A pilot clinical trial of this controller has been done at the LDS Hospital.[60] Figure 4 illustrates typical controller performance. The biggest problem has been the accuracy and dependability of the P_{aO_2} sensor. Until reliable sensors are available, closed-loop control will not become a routine clinical tool.

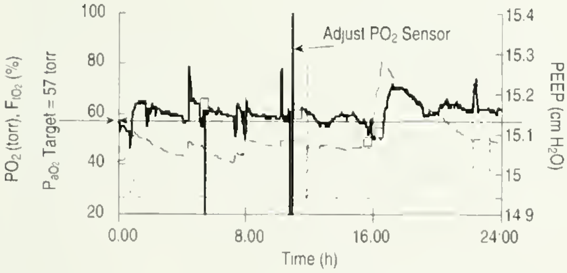

Fig. 4. Typical performance of the closed-loop oxygenation controller at the LDS Hospital in one ARDS patient as part of a small pilot study.[76]

Ethics of Decision Support

A systematic overview of ethical implications of a new therapy requires consideration of four basic principles in biomedical ethics: nonmaleficence (do no harm), beneficence (do good), autonomy (respect patient self-direction), and distributive justice (be fair).[77,78] When the decision to treat a patient is made, and the only choice remaining relates to the technique or process of delivering the treatment, issues usually considered under the purview of the principles of autonomy and distributive justice become less important. The importance of the principles of nonmaleficence and of beneficence remain undiminished. While nonmaleficence is frequently the overriding principle, the commitment of health-care professionals to provide benefit to patients is virtually universal.[77] Striking a reasonable balance between the principles of nonmaleficence and beneficence is a frequent challenge in serving the patient's best interest.[79] In effect, a risk-benefit assessment must be made and is best made with sound data on which to base probability estimates of outcome for the treatment options under consideration. For patients with life-threatening hypoxemic respiratory failure—ARDS, the outcome of interest is patient survival.[80] Unfortunately, credible outcome data are generally unavailable for many medical problems, including ARDS.[81] Only about 15% of medical interventions are supported with any scientific data,[82] and only 0.7% of interventions were reported to be supported by at least moderately strong scientific evidence. Care must be exercised when choosing treatments that have not produced credible scientific outcome data because potent modern interventions can induce harm as well as good. Mechanical ventilation for life-support of ARDS patients has both the potential for benefit[83,84] and the potential for harm.

The principle of autonomy requires patient participation and the acquisition of informed consent for application of new or nonstandard treatments.[77,78] Two opposing arguments can be mounted regarding the need for informed consent for the use of decision support systems with mechanical ventilation. First, these systems may be viewed as new and innovative, nonstandard therapies with undefined risks and benefits. Informed consent would then be mandatory. Second, decision support may be viewed as a tool that merely formalizes and standardizes common practice. The forethought and consensus required for protocol generation[85,86] leads to a more precise and detailed articulation of the explicit and implicit rules applied in standard clinical practice (albeit with variability).[87] Decisions under protocol control would, therefore, be made with more forethought and planning than would decisions made individually by an independent practitioner.[81] Certain iterative therapies, such as mechanical ventilation, can be considered tasks within a single knowledge domain, and, thus, are amenable to decision support. Using this argument, informed consent would not be required because decision support is viewed as an extension of the common practice of generating guidelines,[81] critical paths, and standing orders, all of which are efforts to standardize care. At our institution, the Institutional Review Board has accepted the latter argument, and we do not require that informed consent be obtained for the use of decision support with mechanical ventilation.

The principle of distributive justice raises no obstacles to the use of decision support as long as it is applied to all appropriate patients without prejudice.

The principles of nonmaleficence and beneficence are the source of the major controversy that surrounds protocol-driven care. This controversy is frequently couched in questions such as "How can you be sure the protocol incorporates the best clinical care?" or "Physicians must be free to choose the best therapy for each individual patient." The use of decision support systems challenge the traditional authority of medical experts. Thus, protocol control might be viewed as a threat rather than a complement to the clinical status quo because of the common belief that medical experts are able to maximize patient benefit by individualizing decisions.

Decision support for mechanical ventilation of ARDS patients actually generates decisions that are no less individualized than decisions in many other clinical care domains. Control of clinician response through use of bedside decision support tools is not cookbook medicine. Cookbook medicine is patient invariant medicine with diagnostic or therapeutic interventions fixed and unresponsive to the needs of the individual patient. Many examples of cookbook medicine exist in standard medical practice. The use of a fixed antibiotic treatment regimen for β-streptococcal pharyngitis and the use of standard preoperative orders for postoperative treatment are examples of cookbook medicine and are widely practiced and accepted by the medical profession. Standardization of clinician response through use of decision support only standardizes the response to the patient's expression of the disease. It does not standardize treatment because the patient's treatment (clinical care) depends upon both the clinician's response and the patient's expression of the disease. The patient's expression of the disease is patient specific. There is great variety in the way diseases are expressed in different patients. Therefore, there is great variety in the treatment received by different patients whose care is guided by decision support, even though clinician response is standardized by the use of decision support. This distinction between standardization of clinician response and standardization of treatment appears to be unrecognized by some health care professionals.

Beneficence can involve both the conferring of benefit on the patient and the intent to confer benefit without such achievement. The intention to do good is sometimes persuasive in itself.[77] For the critically ill patient with ARDS in the intensive care unit, for whom the decision to provide mechanical ventilation has already been made, the overriding outcome variable is patient survival. Intentions to do good or to be benevolent pale in importance with actions that achieve increases in survival. Outcome data that can lead to credible estimates of survival for different treatment options become crucial to clinical decision making, whether protocol controlled or not. Unfortunately, necessary data are unavailable and many complex clinical decisions must be based on the clinician's intention to do good. The intention to do good is often insufficient,[88-92] but it is always laudable.[77]

Many ARDS clinical-trial-design flaws and many clinical objections to performing clinical trials for patients with ARDS or other life-threatening conditions are based on ethical concerns originating in the expert or authoritarian paradigm, which has been the foundation of the traditional patient-physician relationship. The physician is the expert and possesses the requisite training, knowledge, and experience to provide the advice necessary to guide the patient towards a favorable outcome. In this process, the patient's *best interest* is served by provision of the *best available therapy*.[93] The physician's belief in the superiority of a therapeutic choice has been cited as fundamental to both ethical decision making[93] and the fiduciary nature of the physician-patient encounter.[79,93] The absence of such a belief in the face of therapeutic options constitutes the state of equipoise—indifference—within an individual physician (individual equipoise) or within the medical community (clinical equipoise).[93,94] As long as equipoise is present, randomized clinical trials comparing therapeutic possibilities seem justified. A number of concerns can be raised in response to this traditional view. First, the implicit assumption that a physician's belief is a reliable reflection of the best available information is violated by our knowledge of general human limitations regarding information processing.[2,57,88,95,96] Ignoring these limitations raises physician belief to a level of undeserved authority. The physician's belief in the superiority of complex therapies may, in fact, undermine the fiduciary relationship that should exist between physician and patient[79,93] by exposing the patient to a less-than-optimal therapy.

Second, the belief of the adherents of a particular policy, in itself, provides no justification for the policy. The belief is nothing more than opinion. The policy can only rationally be justified by data or arguments in evidence. Belief can be based on credible outcome data or on unfounded conjecture, or it may be based on considerations that fall on the continuum between credible outcome data and unfounded conjecture. Belief insufficiently grounded in credible outcome data, even when based on extensive personal and medical community experience, can mislead well-intended clinicians to make decisions that fail to benefit and even risk harm to their patients. The results of the Vineberg procedure[97] for angina pectoris, with its 75% positive placebo effect, and of the Cardiac Arrhythmia Suppression Trial (CAST)[91] with its unexpectedly high mortality associated with effective suppression of premature ventricular beats following myocardial infarction are graphic examples of this danger. Other important examples are easily found and include O_2 therapy for neonatal respiratory distress,[90] gastric freezing for upper gastrointestinal hemorrhage, laetrile for cancer, rapid I.V. infusion of 5-FU for colorectal cancer, intra-arterial infusion of chemotherapeutic agents for colorectal liver metastases, hydrocortisone after myocardial infarction, splenectomy for Gaucher's disease, and frontal lobotomy for the treatment of psychoses. It is of interest, therefore, to find that belief alone has been used to justify some of the most consequential and onerous decisions in critical care medicine.[93,97,98] In the interest of fairness, we acknowledge that some writers demand that physician opinion be based on reliable actuarial data,[89,99,100] but this does not appear to be a universal expectation. That belief itself is neither sufficient nor even always necessary, for the effectuation of correct therapy can be rapidly appreciated. The steadfast belief of the adherents of laetrile therapy did not make the therapy correct, nor could it justify the conduct of a clinical trial.[99] Just as the individual practitioner cannot invoke idiosyncratic good intentions as a defense, so also should this avenue of defense be forbidden to the expert committee that may be charged with defining a standard of care for the medical community. Poorly supported opinion, no matter how well intended, does not become more valid when offered by a dozen experts rather than by one practitioner.

The common clinical concerns raised about decision-supported care are, at least in part, reflections of the strength of the authoritarian paradigm and of the beliefs of the clinicians that practice within this paradigm. Open discussion of the limitations of such beliefs when bereft of supporting scientific data has been an effective means of overcoming these clinical concerns.

Legal Issues of Decision Support

Decision support systems can be a legal shield as well as a legal sword. Practicing under an accepted community standard as prescribed can provide a shield against litigation. If there is a standard, but it is not followed, and if there is no documentation as to the who and why of the departure, the standard becomes a sword that can be effectively wielded in a court of law. To minimize the risk and maximize the legal benefit, we propose the following:

1. Facilitate institutional and departmental approval of decision-support systems as a standard of care.
2. Carefully train staff and monitor the use of the system.
3. If decision-support systems are not used, document the who and why of the decision in the patient's medical record.
4. Provide a method for continuing updates as the system evolves.

In Summary

Decision support for clinicians who care for patients on mechanical ventilation and its effect on reducing inappropriate therapy and variation in practice may have a profound impact on the quality and cost of care. Each of the tools for decision support have strengths and weaknesses. Figure 5 summarizes these tools. In general, as the level of standardization increases, the quality of care increases, the cost of care decreases and the legal liability decreases. Standardization does intrude on caregiver autonomy; however, it is unclear whether this has a positive or negative effect.

Decision support in written or computerized form is not intended to replace the clinician any more than the computers in the F-16 fighter replace the pilot. The computers are used to make the pilot more efficient, and they can do the same for respiratory care practitioners. The primary focus for such systems should be to improve the quality of care. Cost reductions naturally follow improved quality because ineffective therapies are eliminated and length of stay is shortened. The financial benefit of these systems is not in reducing personnel. Our experience indicates that decision support does not result in reduced staffing requirements; however, it does enable existing staff to better care for sicker patients.

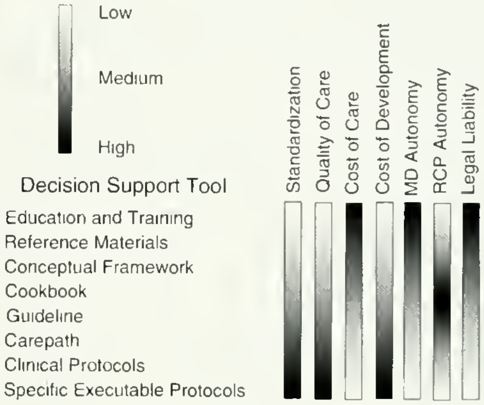

Fig. 5: A summary and ranking of the different decision-support tools with regard to standardization, quality of care, cost of care, cost of development, physician autonomy, respiratory care practitioner autonomy, and legal liability.

The respiratory care profession must avoid producing duplicate and conflicting decision-support tools and adopt standards that allow these tools to be applied effectively throughout the community. Decision-support tools and the standardization of ventilator management as part of an ongoing continuous quality improvement process have the potential for a larger impact than most of the innovations in mechanical ventilation during the last 20 years.

ACKNOWLEDGMENT

Dr East acknowledges the commitment and support of his wife, Katherine, and children, Tommy, Rosemary, and Patrick. Without their loving support and understanding of Dad's long hours, none of this would have been possible.

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East Discussion

> There is incalculable harm done by everyone doing his best.
> — W Edwards Demming

Slutsky: Tom, that was a very interesting presentation. I have a question about your trial. You're doing a trial comparing your computerized system versus standard practice, essentially, but your computerized system is basically a low-stretch hypercapnic strategy; so, which are you really comparing? Are you comparing hypercapnic versus nonhypercapnic? Are you comparing low-stretch versus high-stretch? Or is it the protocol per se that you're testing?

East: The issue is really tough. We grappled a lot with this. A trial to compare protocol versus nonprotocol for the same therapeutic goals and medical philosophies is tough to put together. You basically end up having to control both groups.

Slutsky: If you put your protocolized system into a hospital where the practitioners in that ICU feel strongly about low-stretch, I think that would be at least reasonable. If you put it into a hospital where that's not the case, then you're really comparing apples and oranges.

East: In this trial it is all rolled into one. It's sort of an effectiveness study. The intervention is the protocols. The protocols by their nature embody the medical knowledge. The particular set of medical knowledge we've chosen to put in was one we found to be reasonable. Now, there are others, and you can argue that even if you finish this trial and it looks like things are good, maybe there's a better one. Well, there probably is a better one. This trial really isn't designed to tease out what aspect of the trial is causing the improvement or worsening or whatever. When we're done, we're not going to be able to say that the results are due to making adjustments at regular intervals. All we're going to be able to say is that, as a whole, with the medical knowledge imbedded in this thing, you get this result. You're absolutely right. It's one of the difficulties of doing these trials. One of the issues that the statisticians have brought up to us is, What about cross-over? For instance, as you point out, if you introduce this in an environment in which they're accustomed to using larger tidal volumes and normal pHs, are they going to cross over? Are people going to learn and start to use the treatment philosophies in the protocols, and will your two groups merge? Our experience has been that that's not the case. We collected baseline data for a year at two institutions, so that we knew their baseline practice patterns. We're tracking them over time in the control group to look for cross-over contamination between them.

Slutsky: That sounds like you're aiming at low-stretch, high-stretch, as opposed to protocol versus nonprotocol.

East: Yeah.

Barnes: From your experience, what do you recommend for the respiratory care profession? There's been a tremendous investment in practice guidelines. Is it time to switch and move those guidelines to protocols? An alternative would be to make a bigger effort to determine whether the guidelines actually make a difference.

East: Both are valid approaches. If you put your guidelines out there and try to measure how people use them and how well they use them and what their impact is, I think that would be a good thing to do. My own personal belief is that if you take the guidelines you currently have and build on them—to put something easy to use into the hands of clinicians at the bedside—you're going to have far bigger impact. But that's my bias. Our therapists like this kind of system. They go about their normal job, it gives them advice, the physicians write a standing order, "Treat the patient by protocol." The therapists don't have to track down physicians every time they need to change something on the 'vent.' I realize that may be very different in different institutions. What the policies are—some institutions give big leeway to the therapist in terms of adjusting PEEP and F_{IO_2}. Others do not. But it's been well received by the therapists, I think. They have felt that it's relatively easy to use. The best success has been in groups that already use computers for charting. The hurdle of learning to use computers has already been overcome. Protocols, then, are just a small additional thing. There's no extra learning and no change, really, in the way they practice.

Haas: In assessing the protocols, have you determined whether practitioners have carried out inappropriate orders—bad data entered into the system resulting in an inappropriate order's being carried out?

East: Yes, we have looked at the number of incorrect instructions that were based on bad data or a typo or whatever, and the number that were followed or not, and what the consequences were. What we found was that over 95% of those errors were picked up by the therapist. Because if you type in a P_{O_2} of 400 when it's 40, and it tells you to turn down the F_{IO_2} and/or PEEP, most therapists aren't going to do it. They say, "Oh, yeah. That doesn't make any sense." And they reject it. The ones we found that 'snuck' through were the innocuous ones, like you're in a waiting period, and the instruction says "Keep waiting," when it should have said "Reduce therapy." We found no adverse consequence to patients. But, clearly, the potential's there. Our assumption always is that these are in the hands of therapists who are going to pay attention to what they're doing. It's not a closed-loop system. That's exactly why we have it set up that way.

Haas: I'm glad to hear that because one of the things that I hear often is that people don't like protocols because they are going to take away the assessment ability or skills of the practitioner. As you point out, the software tells you what to do, but you still have to do an assessment to make sure that you were given a correct instruction.

East: You have to do the assessments. We've made the respiratory care group one of our primary participants in the evolution of these protocols because they're the ones on the front line. They can give us the most information about what's working and what isn't working. I think they've appreciated being involved in part of that process.

Slutsky: Tom, do you think in the long-term, if this is introduced over a long period of time, that maybe respiratory therapists and others might lose their edge. You say they now pick up things—"Gee, that doesn't make sense."—In part that doesn't make sense because of the long experience of making many decisions. Is there the danger of therapists becoming just cogs in the system and not think as much about what they are doing? Have you seen that happen at your institution?

East: No. We haven't, but it is a fear and a concern. If you look at some other analogies, for instance, auto-pilots in aircraft and things like that, I don't believe they've seen a big deterioration in pilot performance, but I know in anesthesia they have problems with ... what's the right word—alertness.

Slutsky: Vigilance.

East: And there's some concern that if you have infrequent events—if it's only wrong once every 3 days—that maybe people won't be alert to respond to it. I don't really know. In terms of its impact on teaching, that's another issue that's come up. What do you do with residents and fellows? In our institution, we haven't found it to be a big problem. Most of the residents and fellows come in determined to prove that the computer's wrong, and they spend a great deal of time doing literature searches. We invite that. We say "Hey, great. If you have information that will help us refine these, that's fantastic."

Slutsky: That is more likely to occur at your institution because change in the algorithm can actually occur because you guys are on site and you're excited about it. When the protocol is out into 1,000 other hospitals, that may not be the case.

East: That's right. In our trial, it still is because we take the feedback from all the people at the different sites and incorporate it if it's important. A couple of examples of things we've had to refine are some of our definitions, like "super-satisfactory" oxygenation at sea level ($P_{aO_2} > 110$). We had it defined at 5,000 feet ($P_{aO_2} > 90$). So, we have changed a few things.

Giordano: What approach do you suggest to convince the physicians. I mean, there's an enormous amount of physician resistance to protocols because they fear loss of control over their orders. That's the underpinning of what stops the protocol movement. What's your recommendation there?

East: We have found that physicians in general are pretty receptive if we go to them with data that support that this is going to do a reasonable job of taking care of their patients. It's a lot harder sell if there are no data to support effectiveness. With data, they're more likely to say it's fine. I was really glad that we didn't install Pittsburgh 2 or 3 years ago because they have a lot of capable people, who all have their own opinions. It would have been a lot harder. Now, it has gone relatively smoothly. People are okay about it and have been willing to give it the benefit of the doubt. Now that doesn't mean the first time it does something 'screwy' they won't 'jump down our throat.' But, in general, it's worked out okay. Clearly, that's one of the battles. One of the things we've done is to do some hand holding with physicians, and we provide 800-number call support 24 h/day. If they have questions or problems they can call us and talk about them. If they don't want to do it, they don't have to. They can say they don't agree with the protocol and not follow it.

Giordano: Do you advise those who want to institute protocols to develop a pilot project and then use the results of that effort, then, to sell the concept?

East: Right. I think that would be a good way to approach it, but I think you're going to need (if you're developing protocols in-house and are not bringing them in from somewhere else) some physician advocates who think it's a good idea to start with. Otherwise, you may never even get the pilot data. But if you can get a physician advocate to talk to his colleagues and say, "Hey, give us a chance. Let us do 20 patients and see what happens."

Kacmarek: I think the other thing that will change those attitudes is the change in health-care financing. More institutions and practitioners will tolerate well-designed decision-making systems if they are proven to be cost saving. We're looking at a 30% reduction in our residency programs over the next few years. That kind of reduction is going to have an impact on the physician's ability to do the day-to-day management at all levels. To be able to unload this kind of decision making to other practitioners via well-accepted protocols is going to become increasingly more acceptable.

Peruzzi: Three things that play into this come to my mind immediately, as far as physician cooperation goes. First, I think that people are more likely to be supportive if they feel that they have some input into the protocols and that there can be modifications for individual institutions. Second, I think that people will feel more comfortable if there are bypass options, if there are clinical circumstances in the standard protocol about which they don't feel comfortable, they can step out of the protocol and effect whatever changes they feel are appropriate in that circumstance. Third, I think that the degree of objection is going to be directly proportional to the amount of time people have to object. As finances change and primary care becomes the focus, as anesthesia house staff and surgery house staff are reduced, fewer and fewer people are going to be interested in objecting, being called, and having that control because their time is going to be constrained doing other things. They're going to be more supportive of something that runs on its own with only periodic alerts when things go awry.

East: One thing that we have seen when we go in to a new institution is that it's not uncommon for the physicians to write an order, "Well, follow the protocol, but if the PEEP goes above 15, call me, or if it changes by more than x amount, call me." Then as time goes on they relax that somewhat. I think it's up to individual physicians, too—their own personal bias. The other thing that we have found that kind of eases the pain a bit is that we've taken clinicians who have actually used them to the new facilities for clinician-to-clinician discussion. Terry Clemmer, our ICU director, has done some of this. Because the feeling is that they will get a straight answer from other clinicians as opposed to the folks who are pushing it for the research project. They can discuss the pros and cons and the problems that come up. The biggest supporters in our institution, at least from a physician-group perspective, are the private practice pulmonary docs. They love it. They say they can manage more patients in less time than they ever could before. In fact, we tried to turn the protocols off after the $ECCO_2R$ trial ended, and they refused. They said that they couldn't imagine going back to managing patients without it.

Adams: There will eventually be poor outcomes. Have you discussed the legal liabilities of poor outcomes?

East: Yes, I didn't bring slides on the legal aspects, but they're there for therapist-driven-protocols as well as for guidelines. You need to get approval in your facility as a standard of care for whatever protocol you want to use. Otherwise, they're going to hang you for not following the standard of care in your own institution. Once it's approved as a standard of care in your institution, then if you deviate from it you need to document why and who did it. That's part of the audit trail in our computerized protocols. Michael Meehan[1], counsel for the Cleveland Clinics, has described problems with therapist-driven protocols, if this information is not documented and you are taken to court. They put the protocol up and say "Well, did you follow it?" When you say "No" and there's no reason documented for why, you've lost the case, basically.

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New editions of the following textbooks are available from the publisher.

Manual of Pulmonary Function Testing, 6th Edition, by GE Ruppel. Soft-cover, 507 pages, illustrated. St Louis: Mosby-Year Book Inc, 1994. $29.95.

Clinical Application of Blood Gases, 5th Edition, by BA Shapiro, WT Peruzzi, R Templin. Hardcover, 427 pages, illustrated. St Louis: Mosby-Year Book Inc, 1994. $41.95.

Egan's Fundamentals of Respiratory Care, 6th Edition, by CL Scanlan, CB Spearman, RL Sheldon. Hardcover, 1,197 pages, illustrated. St Louis: Mosby-Year Book Inc, 1995. $61.95.

Résumés and Personal Statements for Health Professionals, by JW Tysinger. Softcover, 196 pages, illustrated. Tucson AZ: Galen Press LTD, 1994. $15.95.

Dr Tysinger has been a health education program advisor for more than 20 years, spending 9 of those associated with medical school. According to the author, **Résumés and Personal Statements** was written to fill a void in the resources for constructing résumés and personal statements. We believe no such void exists. How-to texts, articles, and manuals on writing a résumé are not hard to locate. This particular text is, however, targeted to students and entry-level health professionals and contains examples of résumés and personal statements for various allied health, nursing, and medical specialties.

Of the 30 résumé examples, 14 are for medical school, residency program, and first-job-after-residency applicants. Lest the respiratory therapist expect a stellar template to follow, there is only one example for a respiratory care practitioner (RCP) in the résumé section, and none in the section that includes examples of personal statements. This RCP example for a CRTT uses incorrect credentials for a registered respiratory therapist (RT). Like many of the examples with celebrity names (Don Shula, E Allen Poe, Gertrude Stein)—the respiratory therapist is Paul Shafer. (It is interesting that this RCP's hobbies include trumpet and jazz.) Further, Paul's respiratory therapy diploma seems to be overshadowed by his associate degree from an emergency medical technology (EMT) program, and a bachelor of arts degree in music.

To Tysinger's credit, the book includes a "Personal Experience Inventory" to be completed before progressing through the book; this is very user-friendly and could help an individual to organize thoughts and experiences. The résumé examples and 28 personal statements illustrate good ideas and different ways to organize and present information. Tysinger also includes a personal marketing inventory (for use when writing a personal statement) to help students and first-time job seekers identify school-based and experience-based activities that have shaped their lives.

The book is logically organized into 12 chapters, beginning with the importance of constructing a good résumé and personal statement and ending with tips for building these over the user's professional career. The tips offer good advice about becoming active in professional organizations and volunteer opportunities. In a step-by-step process for the résumé and personal statement, Tysinger takes the reader through a definition of the topic; the personal marketing inventory; a plan for construction; and many examples. One résumé and personal statement is corrected; this is helpful because many of the mistakes are common errors. Tysinger encourages the reader to make statements specific to the individual or institution rather than simply inserting the name of an institution or a person into a standard form letter. The text also includes a short chapter (5 pages) on how to construct cover letters and thank-you notes.

The book is probably best suited for students and first-time job seekers. It is logically organized, easy to use, and presents material in a way that is not overwhelming to the reader. The only section that is unique to health care (other than the examples used) is one that helps the user to identify clinical and professional areas of interest. Tysinger includes a tear out "Feedback Form" designed for readers to send examples for the next edition or comments on their likes and dislikes. Needless to say, we asked for a new and improved Paul Shafer in the next edition.

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Just Say No to Therapeutic Touch!

In her response to my letter,[1] published in the February issue of the Journal, Ms Haines seems somehow to have misinterpreted my letter opposing Therapeutic Touch (TT). I am fully aware that Krieger[2] and Quinn[3] are proponents of TT, but to state that Meehan's study[4] supports TT is simply not true! The following is taken directly from the abstract accompanying that article.

> ... The hypothesis, that TT would significantly decrease postoperative pain compared to the placebo control intervention, was not supported.

Those are the words of the author and require no further comment on my part.

Ms Haines then restates that her references support TT. Well, actually some of them do and some of them do not. I will address the issue of the references that do support TT later in this letter but first the articles that do not.

Her reference to other work by Meehan supposedly demonstrates that there would be a greater decrease in postoperative pain in patients given TT than in those given Mimic Treatment (placebo). The abstract, however, states the following:[5]

> ... It was hypothesized that there would be a greater decrease in post-test acute pain scores in subjects treated by TT than subjects treated by Mimic Treatment. ... Although a decrease was found in post-test acute pain scores of subjects treated by TT compared with subjects treated by Mimic Treatment, it was not great enough to be significant at the .05 level. The hypothesis, therefore, was not supported. Standard Treatment set a sensitive internal standard, but was significantly more effective than TT.

In other words, TT was significantly worse than standard nursing care with no special interventions in reducing pain in postoperative patients!

The reference by Ms Haines to the article by Quinn[3] that supposedly supports TT actually does not. The abstract states the following:

> This study replicated and extended previous published research which suggests that TT involves an energy exchange. The theorem that eye and facial contact between TT practitioners and subjects should not be necessary to produce the effect of anxiety reduction was deduced from the Rogerian conceptual system and tested. *This theorem was not supported.* [My emphasis.] Numerous explanations for failure of the hypotheses to be supported are posited, among them the impact of the research design, effects related to the investigator as practitioner, and medication. The influence of medication in particular is so pervasive in the sample that it would seem to be the most reasonable explanation.

Major problems with the Quinn article[3] as pointed out by other authors include:

1. When the results did not support the initial hypothesis, the author shifted the search for positive results and reported significant effects on hypertension not predicted in the study design. However, the author did perform proper randomization in this study, therefore the effects of medication should have been eliminated as a confounding variable.[6]

2. The investigator performed the interventions which can potentially introduce bias.[7]

The rest of the references cited by Ms Haines either have serious methodologic flaws that invalidate the results or they could not be replicated in studies of the same type. If the results of a study cannot be reproduced then those results may have simply been due to chance.

The article by Keller and Bzdek[8] has been criticized for the following reasons:

1. The investigators administered the interventions.[4]
2. Placebo control was inadequate.[9]
3. Results could not be reproduced.[4]
4. No explanation was made regarding why a 4-hour time interval between intervention and post-test was chosen.[10]
5. 50% of the placebo group in the 4-hour test interval were removed from data analysis, producing a level of statistical significance not present when they were included.[10]
6. This article is used in a textbook on critical thinking[11] as a probable example of "post hoc ergo propter hoc" (fallacy of false cause) reasoning. (I highly recommend this text for anyone interested in improving their critical thinking skills.)

The article by Heidt[12] has been criticized for the following reasons:

1. No randomization of subjects was performed.[10]
2. The primary investigator administered both interventions of TT and casual touch (placebo).[4]
3. The tool used to measure anxiety level was of questionable validity.[10]

The second article written by Quinn[13] and referred to by Ms Haines has its share of problems also. These include:

1. The results could not be replicated by the same author in a later study.[3]
2. Other studies have shown no decrease in anxiety.[7]

The last reference of Ms Haines is to an article by Wirth et al.[14] The main author could not replicate the results using the same research mode.[15] The abstract of this follow-up study states:[15]

> ... Results showed significance for the treated versus the control group but *in the opposite direction from that expected.* [My emphasis.]

The author then goes on to give possible explanations for the negative results but overlooks the more likely explanation that TT does not work! A second problem with the article referenced by Ms Haines is that according to Meehan:[16]

> ... In studies that purport to link TT to wound healing, it clearly is not TT that is being tested.

I believe that the final score on the six references provided by Ms Haines is:

1. Two did not support the authors' primary hypothesis.
2. Four had serious methodologic flaws or were unable to be replicated.

In a letter to the editor of the *American Journal of Nursing*,[16] Meehan described the current evidence supporting TT:

> … anecdotal evidence is not enough. They bring to the fore, yet offer no hope of answering, the two most serious charges leveled against proponents of TT: the practice-related charge of quackery or witchcraft (in kinder terms, faith-healing), and the academic charge of pseudoscience.
>
> The first charge is understandable, given the nature of TT and the dramatic therapeutic outcomes attributed to it. What is needed from proponents are rigorous, precise, and critical analyses of the concepts and assumptions underlying TT. Reports of practice must be presented from the perspective of a disinterested observer, and should include carefully documented outcomes and critical evaluation … proponents would do well to avoid trivializing TT by associating it with 'new age' ideas and organizations.
>
> … The second charge of pseudoscience strikes at the heart of nurses' intellectual and scientific integrity and, sadly, is not unfounded.
>
> … What current research about TT tells us, according to Popper's principle of refutation and verification is that *there is no convincing evidence* [my emphasis] that TT promotes relaxation and decreases anxiety beyond a placebo response, that the effects of TT on pain are unclear and replication studies are needed before any conclusions are drawn. Other claims about outcomes are, in fact, speculation. In studies that purport to link TT to wound healing, it clearly is not TT that is being tested.

Readers should note that this is the same Meehan that Ms Haines refers to as supporting TT!

Ms Haines states in her response to my letter that she listed "just six of the clinical research studies." The implication is that there is a huge volume of evidence supporting TT. Let's take a closer look at the rest of the literature.

In 1992, Quinn of the University of Colorado (CU) was directed by the State Board of Nursing to document the efficacy of TT. Quinn produced a "reading list" of 201 references. This list was analyzed by an organization of concerned citizens known as the Rocky Mountain Skeptics. Their findings are:[17]

1. Eight of the references were duplicates or separate listings of different parts of the same work. The total should have been 193, not 201.
2. Ten were to articles published in the popular press.
3. Forty-four articles were classified as 'popular' works, incidental mentions, and unrelated publications. They were not research articles and cannot be used to defend TT.
4. Twenty-eight articles were in journals with little, no, or questionable reputations.
5. Thirty-eight were nonpeer-reviewed book appearances. Three of these are videotapes by Quinn.
6. Two are correspondence responding to attacks.
7. Six are dissertations and theses that may be oblique to the topic.
8. Three are reviews (no original work).
9. Two are probable reviews (no original work).
10. Five are supportive dissertations and theses. The abstracts reveal uniformly small samples and poor experimental designs.
11. Five are possibly authentic research papers. The publications are obscure and may not be refereed journals.
12. Six are genuine research articles. All but one of these has been attacked by others for various reasons.
13. Thirty are unrefereed articles in newsletters and trade publications.
14. Twelve are contrarian responses. Some of these 12 include dissertations that attempt, but fail, to confirm the TT hypothesis. The others are not marked as being skeptical of TT.

In summary, the "evidence" supporting TT provided by one of its leading advocates, appears to crumble upon close examination.

The Rocky Mountain Skeptics took their findings and concerns about public funding of pseudoscience directly to the Board of Regents (BOR) of CU.[18] The BOR appointed a commission under Dr Henry Claman to investigate the scientific credibility of TT. The findings of the Claman Commission include the following:[19]

> … To date, there is not a sufficient body of data, both in quality and quantity, to establish TT as a unique and efficacious healing modality. … This lack of data and consequently the perceived uncertainties … brings the potential to have a negative effect and stature and reputation of the School of Nursing. Qualitative judgments and evaluations are not sufficient to document and establish TT as an efficacious therapeutic or healing modality. … If an effect is observable, it can be measured. It is not adequate to state that TT involves mechanisms which cannot be proven by ordinary methods. Such comments are a disservice to science and the practice of healing and demonstrates a commitment to metaphysics and the mystical view of life rather than to a scientific or rational view of life. … It is inappropriate in the context of a health sciences center to teach and practice TT for another 20 years in the absence of validation of TT as an efficacious healing modality.

I agree with Ms Haines on one point. It is up to each individual to investigate the validity of TT. Then an informed choice can be made whether to incorporate it into patient care. The choice comes down to this question: Should we incorporate an unproven modality that has its foundations in Eastern mysticism, the occult, and 'new age' tenet, or should we insist upon evidence based on the scientific method of performing research? I think the choice is obvious.

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I am greatly indebted to Linda Rosa RN BA BSN Corresponding Secretary of the Front Range Skeptics and Co-Chair of the Questionable Nurse Practices Task Force of the National Council Against Health Fraud (NCAHF) for providing me with most of this information.

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Humidity & Standards: More Questions than Answers

In the January issue of the Journal, Miyao et al[1] describe the effects of different levels of humidity in inspired gases on the hydration of simulated pulmonary secretions. The authors have made similar observations in previous publications.[2,3] I find the concept that the authors introduce related to the cross-sectional area of the airway and secretion drying an interesting one. Briefly, Miyao et al suggest that as the cross-section of the airway and, consequently, surface area increase, the ability of gases with low relative humidity to cause inspissation of secretions is reduced. Conversely, gases with low relative humidity delivered to the endotracheal tube cause accelerated drying of secretions due to a small cross-sectional area. This theory deserves some consideration.

I have rarely seen obstruction of an artificial airway secondary to insufficient humidification when a heated humidifier and an unheated wire circuit set to deliver 32° C at the Y-piece are used.[4] Yet, when heated wire circuits with a large temperature offset are used, there appears to be an increased incidence of airway obstruction.[2,3,5] These observations, when considered together, are puzzling. If a gas at 32° C and 100% relative humidity, or RH%, (absolute humidity, AH = 34 mg H_2O/L) is warmed to body temperature (37° C at 100% RH = 44 mg H_2O/L), then the contribution of the respiratory tree is 10 mg H_2O/L. Similarly, a gas delivered to the airway at 37° C at 75% RH (AH = 33 mg H_2O/L), warmed to body temperature, requires the respiratory tree to add 11 mg H_2O/L. Yet, inspissation of secretions is rarely seen in the former and is more frequently reported in the latter. The answer may be explained by the concept relating cross-sectional area to moisture loss. That is, gas with low RH% entering the artificial airway has a concentrated drying effect in the large airways resulting in thick secretions. Gas at a high RH% but lower AH has a less immediate effect and slowly increases temperature and water content from a larger heat sink (increasing cross-sectional area of the respiratory tree.) Miyao et al's paper is a valuable addition to the literature, if only this concept receives more attention.

That being said, there are limitations to Miyao et al's work. I believe that their model of an artificial airway and respiratory tree is simplistic. The model uses only unidirectional gas flow to simulate the bidirectional flow present in the respiratory tract. Therefore, the retention of moisture that occurs during expiration, an important portion of normal respiratory tract function, is missing from the model. The authors use dry filter paper to simulate the endotracheal tube; whereas, most thermolabile artificial airways allow some heat transfer to inspired gases from the moist respiratory mucosa and can act as inefficient heat and moisture exchangers.[6] Clearly, the model used in this study can be characterized as a poor representation of the real thing. Of course, the authors have chosen simplicity to minimize interaction of variables and provide tighter control of delivered humidity and temperature, but I'm afraid that they may have simplified the model to the extent that it no longer represents the in vivo characteristics of the system they intended to study. In fact, I suggest that the results obtained with their simple model were predictable. To put it simply—a dry gas will extract moisture from a gel and a wet gas will add moisture to a gel. What other possibilities are there?

Another technical point that needs to be settled—the difference in temperature between the humidification chamber and the airway temperature probe. Manufacturers have allowed an offset of as much as 5° C so that humidifiers can be used to deliver warm gases to patients without an artificial airway. Using the previous example, an airway temperature of 37° C allows an AH of 44 mg H_2O/L. If the chamber temperature is set 5° C below airway temperature, the AH of gases leaving the chamber is 34 mg H_2O/L, and when the gases reach 37° C the relative humidity is 75%. This situation should never occur in a patient with an artificial airway. Unfortunately, clinicians sometimes adopt this unsafe practice to minimize condensate within a heated wire circuit. Although this practice may make life simpler for the respiratory care practitioner, it should be eradicated. Blame does not lie with the manufacturers in this instance, but with the caregivers.

Lastly, what about the standard? ASTM currently recommends that 30 mg H_2O be delivered to the end of the "breathing tube."[7] If one looks in the Appendix of that standard, one finds the definition of breathing tube—basically, the tube that connects to the Y-piece. So, the standard says, heated humidifiers should provide a minimum of 30 mg H_2O/L at the end of the breathing tube. Another point about ASTM standards—they are written so that manufacturers can use like test methods to prove the safety and efficacy of their devices. They are not intended to be standards of clinical practice or recommendations for optimal practice. Those can be found in the AARC Clinical Practice Guidelines.[8]

In the end, Miyao et al leave us with the nagging humidification question. What is the optimum level of humidity that should be delivered to patients with an artificial airway? Do we try to provide the optimum, minimum safe, or maximum humidity? How do we measure the effects of humidity strategies? Do we look at cellular structure, tracheal mucus-flow velocity, ciliary structure, secretion volume, secretion viscosity, or simply patient response (eg, the presence of a plugged vs an unplugged endotracheal tube)? Well-designed animal and clinical research should be directed toward answering these questions.

Finally, assuming the prerogative of the letter writer, I will muddy the issue by espousing my own ideas. Optimum humidification is a time-, environment-, and condition-related answer. By way of explanation, a patient with normal lungs, entering the operating room for a 2-hour procedure probably requires minimum humidification. The moisture from the circle absorber appears to be sufficient, if for no other reason than the empirical evidence. Everyday in this country, thousands of people with normal lungs have operations, are intubated, receive only the humidity provided by the circle absorber, wake up, and go home none the worse for wear. On the opposite side of the spectrum, the patient with COPD, excessive secretion production, fever, and minute ventilation of > 10 L/min who requires invasive mechanical ventilation needs heated humidification at 33-35° C with 100% RH. In between are a host of patients for whom—depending upon the length of time the airway is bypassed, the previous lung health of the patient, and the desired humidification effect (optimum, minimum, maximum)—the required humidity and humidification devices are different. This is one of the reasons the Mechanical Ventilation Consensus Conference recommended 25-35 mg H_2O/L.[9] Because a previously lung-healthy patient ventilated in the recovery room for 2 hours after surgery requires only an artificial nose that delivers 25-26 mg H_2O/L. However, a patient with COPD, copious secretions from necrotizing pneumonia, and temperature of 39° C requires heated humidification and humidity approaching alveolar conditions. So, unfortunately, as usual, patients do not lend themselves to being easily pigeon-holed. Perhaps the real goal of humidification is "appropriate humidity" or that which is necessary for the patient based on setting, lung health, length of use, and desired effect. What is the appropriate level? I don't know. I do know, however, that the appropriate level changes from patient to patient and is best judged by careful patient assessment by the respiratory care practitioner.

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Dr Miyao and colleagues respond:

We appreciate Mr Branson's interest in our paper[1] and are glad that he had such a good understanding of the paper's point. We would like to respond to the points he raised.

We agree with the statement that a gas at 32° C and 100% RH (AH = 34 mg H_2O/L) reduces the incidence of airway obstruction more than a gas at 37° C and 75% RH (AH = 33 mg H_2O/L). This is because the deficit of 11 mg H_2O/L (alveolar gas AH of 44 mg H_2O/L − 33 mg H_2O/L) would be taken mainly from the upper airway in the latter gas; whereas, the former would derive 11 mg H_2O/L from the periphery.

We readily admit that a one-way flow of gases, as was used in the model, does not mimic physiologic conditions. Our first reason for adopting unidirectional flow has to do with inspissation of secretions. Under actual conditions, expiration acts to hydrate secretions that have been dried during the inspiratory phase. However, inspissation of secretions would never occur even after long-term exposure to a dry gas if such a countercurrent system were completely satisfactory, and humidifiers would not be necessary. Inspissation of secretions, therefore, is caused by the characteristics of the inspired gas. The second reason has to do with temperature control. No attempt was made to simulate actual temperature conditions in the model because an exact simulation can be obtained only by leaving the upper part of the model at room temperature, the middle part at oral temperature, and the lower part at body temperature. If bidirectional flow had been adopted in our model, it would have been necessary to prepare a lung model complex in regard to temperature and moisture. The experiment would have been difficult to carry out and the results difficult to interpret. The third reason stems from a weakness in the available technology. The response time of even the most recently developed hygrometer, such as was used in the present study, is too slow to respond adequately to ordinary respiration.

In summary, we chose to simplify the experiment by using one-way flow in order (1) to save time by shortening the overall time required for exposure of the gel, (2) to eliminate the complex effects of expired gas, and (3) to avoid technical problems. We think that in-vivo studies should follow.

In our opinion, Mr Branson oversimplified the experiment, in writing "To put it simply—a dry gas will extract moisture from a gel and a wet gas will add moisture to a gel." But what is a dry gas? What is a wet gas? Which gas used in our study is drier, Gas 3 (AH = 30.5 mg H_2O/L, 38° C, 66% RH) or Gas 5 (AH = 27.2 mg H_2O/L, 28° C, 100% RH)? Although Gas 5 would derive more water from the whole lung than Gas 3, Gas 5 could add more moisture to the upper airway than Gas 3. Therefore, in the upper airway, Gas 3 may be drier than Gas 5, yet in the whole lung Gas 5 may be drier than Gas 3 (see detail in the figure from Reference 1, reprinted here). Absolute humidity is essential for determining the total amount of water derived from the patient's lung, and relative humidity is decisive in determining whence most of the water is derived.[2]

Mr Branson also mentioned that a humidifier with a heating wire can be used on patients without an artificial airway. But, one of the main reasons for using a heating wire is to eliminate the condensate from the circuit. We don't think it is necessary to heat airway temperature 5° C higher than the temperature of the humidification chamber to eliminate condensate. Who would request such a setting? Is there any pathologic condition in which such a setting might be needed? Although excessive condensate should be avoided to lessen the likelihood of nosocomial infection, a temperature difference of 0° C between the airway and humidification chamber is enough to eliminate excessive condensate.

We regret that many intensivists, ICU nurses, manufacturers, and, especially, the ISO and ANSI Committee seem to have little interest in relative humidity. We don't want ANSI to specify the standards of clinical practice or make recommendations for optimal practice, but we do want a standard of minimum safety. We may safely say that AH should be greater than 30 mg H_2O/L by adding the condition that RH be over 95%. This is a simple request. Our study was not designed to indicate optimal values of AH and RH. We hope that in vivo studies will follow. We also hope that this study will arouse interest in relative humidity.

The writer stated "...a patient with normal lungs, entering the operating room for a 2-hour procedure probably requires minimum humidification. ..." This may be forgiven in a patient with normal lungs and for a short procedure. Such patients have a reserve against bad conditions. Delivery of dry gases should be avoided for even a wet lung as in COPD. The more the secretions, the more easily they coagulate in an artificial airway when a dry gas is delivered. We don't agree that the appropriate level of the humidity changes from patient to patient. We do believe that RH should be near 100% with AH above 30 mg H_2O/L. The writer mentioned an artificial nose that delivers 25-26 mg H_2O/L without many complications. This is probably because RH is high enough to prevent the drying of secretions in the artificial airway. We may safely say that 100% RH is a minimum safety standard.

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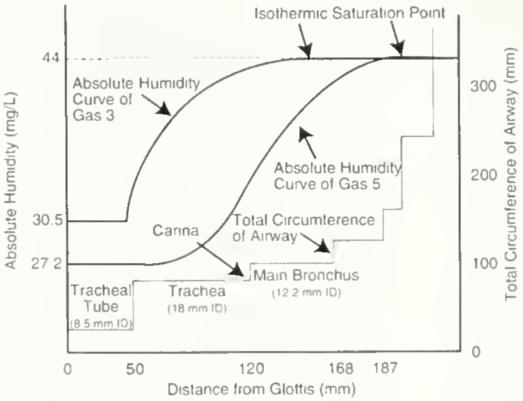
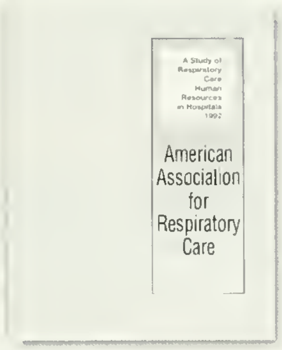
2. Miyao H, Hirokawa T, Miyasaka K, Kawazoe T. Relative humidity, not absolute humidity, is of great importance when using a humidifier with a heating wire. Crit Care Med 1992;20(5):674-679.



Possible absolute humidity (AH) curve of Gas 3 and Gas 5 in inspiratory phase and total circumference of airway. Due to reduced relative humidity, Gas 3 (38° C, 66%, 30.5 mg/L) has greater ability to dehydrate mucosa than Gas 5 (28° C, 100%, 27.2 mg/L). Gas 3 would deprive the trachea of the greatest amount of water, resulting in steeper rise in AH in this area. AH increases because water is supplied from tracheal mucosa. Mucosal surface area, which is not proportional to total cross-section of airway but to total circumference, rapidly expands from carina. Steplike line represents total circumference of airway (calculated by multiplying diameter × π × airway generation number) adapted from data in Weibel's work,[22,23] which shows the average diameters and lengths of Human Adult Airway Model "A." Right side of vertical axis refers to total circumference of airway in mm. Horizontal axis refers to distance from glottis in mm. Mucosal surface area is calculated by multiplying circumference by distance; mucosal surface area can be obtained by integrating area below line of total circumference of airway. If speculation about shape of AH curve for each gas is correct, amount of water deprived per unit area of tracheal mucosa by Gas 3 would be larger than that by Gas 5, although total amount of water deprived by Gas 3 (13.5 mg/L = 44 – 30.5) is actually smaller than that deprived by Gas 5 (16.8 mg/L = 44 – 27.2). (Reprinted from Reference 1.)

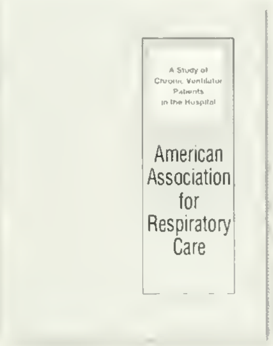
The AARC Human Resources Survey: A Study of Respiratory Care Human Resources in Hospitals


A Study of Respiratory Care Human Resources in Hospitals 1992
American Association for Respiratory Care

Covers a wide range of human resource issues, including compensation, numbers of full-time equivalents, job vacancy rates, education, credentialing, and licensure. Even includes information on age, sex, and years of experience. Includes comprehensive summary, position profiles, salaries, education, experience, credentials, and regional demographics. Vacancies are inventoried. 68 pages, 66 tables.

Item BK12 $35 ($50 nonmembers)

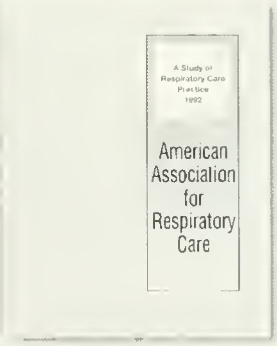
A Study of Chronic Ventilator Patients in the Hospital


A Study of Chronic Ventilator Patients in the Hospital
American Association for Respiratory Care

Chronic ventilator-dependent patients are costing American hospitals more than $9 million per day according to this Gallup study conducted for the AARC. This important study provides information on patients who depend on life-support systems; why, how, and where they are being treated; and the cost of treatment. 47 pages, 9 tables, 12 figures.

Item BK20 $25 ($50 nonmembers)

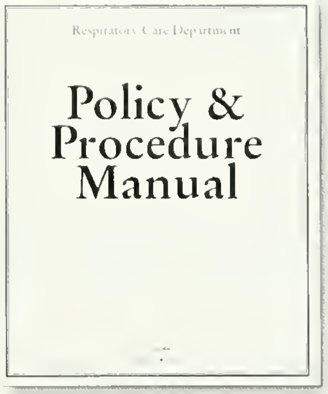
A Study of Respiratory Care Practice


A Study of Respiratory Care Practice 1992
American Association for Respiratory Care

This study examines the practice of respiratory care in today's health care environment and how hospital reorganization is affecting the profession. Includes chapters on medical direction, current respiratory care services, nontraditional services, respiratory care protocols, and hospital service reorganization. 38 pages, 34 tables, 15 charts.

Item BK17 $20 ($40 nonmembers)

Policy and Procedure Manual



Save time and money by making your department more efficient with the *Policy and Procedure Manual*. Its 130 pages of policies and procedures cover the aspects of administrative and clinical respiratory care for both adult and pediatric practice. Sections on administrative policies, therapeutics, clinical monitoring, and mechanical ventilation. Standardized formats include objectives, indications, equipment, policies, contraindications, troubleshooting, procedures, hazards, and assessment of effectiveness.

Item BK6 $60 ($70 nonmembers)

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Item BK7 $9 ($12 nonmembers)

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Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

The Lambda Beta Society

The National Honor Society for the Profession of Respiratory Care

For information about the Lambda Beta Society—
the National Honor Society for the
Profession of Respiratory Care,
contact the Society Office at
1701 W Euless Blvd, Suite 200, Euless TX 76040
(817)283-4269.

Call for Abstracts

1996 RESPIRATORY CARE OPEN FORUM

Deadline: April 28, 1996

Accepted abstracts will be printed in the October 1996 issue of RESPIRATORY CARE

Selected authors will present their research at the OPEN FORUM during the AARC 42nd International Convention & Exhibition in San Diego, California November 3-6, 1996

The National Board for Respiratory Care—1996 Examination Dates and Fees

Examination	Examination Date	Examination Fee	
CRTT Examination	March 9, 1996 Application Deadline: January 1, 1996	$ 90 60	(new applicant) (reapplicant)
	July 20, 1996 Application Deadline: May 1, 1996	100 60	(new applicant) (reapplicant)
	November 9, 1996 Application Deadline: September 1, 1996	100 60	(new applicant) (reapplicant)
RRT Examination	June 1, 1996 Application Deadline: February 1, 1996	100 60	Written only (new applicant) Written only (reapplicant)
		110	CSE only (all applicants)
	December 7, 1996 Application Deadline: August 1, 1996	210 170	Both (new applicant) Both (reapplicant)
CPFT Examination	June 1, 1996 Application Deadline: April 1, 1996	110 80	(new applicant) (reapplicant)
RPFT Examination	December 7, 1996 Application Deadline: September 1, 1996	160 130	(new applicant) (reapplicant)
Perinatal/Pediatric Respiratory Care	March 9, 1996 Application Deadline: November 1, 1995	160 130	(new applicant) (reapplicant)

For information about other services or fees, write to the
National Board for Respiratory Care, 8310 Nieman Road, Lenexa KS 66214, or call (913) 599-4200.

1996 Call for Abstracts

RESPIRATORY CARE • OPEN FORUM

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the OPEN FORUM during the AARC International Convention & Exhibition in San Diego, California, November 3-6, 1996. Accepted abstracts will be published in the October 1996 issue of RESPIRATORY CARE. Membership in the AARC is not required for participation.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report **(1) an original study, (2) the evaluation of a method or device, or (3) a case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, or health-care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a poster at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

Essential Content Elements

Original study. Abstract *must* include (1) Background: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

Method/device evaluation. Abstract *must* include (1) Background: identification of the method or device and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

Case report. Abstract *must* report a case that is uncommon or of exceptional educational value and must include (1) Introduction: Relevant basic information important to understanding the case. (2) Case Summary: Patient data and response, details of interventions. (3) Discussion: Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

Accepted abstracts will be photographed. The first line of the abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location. Underline presenter's name. Type or electronically print the abstract *single spaced in the space provided on the abstract blank.* Insert only one letter space between sentences. Text submission on diskette is encouraged but must be accompanied by a hard copy. Identifiers will be masked (blinded) for review. Make the abstract all one paragraph. Data may be submitted in table form, *and simple figures may be included provided they fit within the space allotted.* No figures, illustrations, or tables are to be attached to the abstract form. Provide all author information requested at the bottom of abstract form. A clear photocopy of the abstract form may be used. Standard abbreviations may be employed without explanation. A new or infrequently used abbreviation should be preceded by the spelled-out term the first time it is used. Any recurring phrase or expression may be abbreviated, if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (214) 243-2272.

Deadline Allowing Revision

Authors may choose to submit abstracts early. Abstracts postmarked by February 11, 1996 will be reviewed and the authors notified by letter only to be mailed by March 22, 1996. Rejected abstracts will be accompanied by a written critique that should, in many cases, enable authors to revise their abstracts and resubmit them by the final deadline (April 28, 1996).

Final Deadline

The mandatory Final Deadline is April 28 (postmark). Authors will be notified of acceptance or rejection *by letter only.* These letters will be mailed by July 15, 1996.

Mailing Instructions

Mail (Do not fax!) 2 clear copies of the completed abstract form, diskette (if possible), and a stamped, self-addressed postcard (for notice of receipt) to:

RESPIRATORY CARE OPEN FORUM
11030 Ables Lane
Dallas TX 75229-4593

1996 RESPIRATORY CARE OPEN FORUM

Abstract Form

18.8 cm or 7.4"

13.9 cm or 5.5"

1. Title must be in all upper case (capital) letters, authors' full names and text in upper and lower case.
2. Follow title with all authors' names including credentials (underline presenter's name), institution, and location.
3. Do not justify (ie, leave a 'ragged' right margin).
4. **Do not use type size less than 10 points.**
5. All text, tables, and figures must fit into the rectangle shown.
6. Submit 2 clean copies. This form may be photocopied if multiple abstracts are to be submitted.

Mail original &
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paid postcard) to

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Early deadline is
February 11, 1996
(postmark)

Final deadline is
April 28, 1996
(postmark)

Presenter

Name & Credentials

Mailing Address

Voice Phone & Fax

Corresponding Author if Different from Presenter

Name & Credentials

Mailing Address

Voice Phone & Fax

MEDWATCH

THE FDA MEDICAL PRODUCTS REPORTING PROGRAM

For VOLUNTARY reporting by health professionals of adverse events and product problems

Page ___ of ___

Form Approved OMB No 0910-0291 Expires 1/31/96
See OMB statement on reverse

FDA Use Only (RESP CARE)

Triage unit sequence #

A. Patient information

1 **Patient identifier**
In confidence

2. **Age at time of event:**
or
Date of birth:

3 **Sex**
☐ female
☐ male

4 **Weight**
___ lbs
or
___ kgs

B. Adverse event or product problem

1. ☐ **Adverse event** and/or ☐ **Product problem** (e g , defects/malfunctions)

2. **Outcomes attributed to adverse event** (check all that apply)

- ☐ death ______ (mo/day/yr)
- ☐ life-threatening
- ☐ hospitalization – initial or prolonged
- ☐ disability
- ☐ congenital anomaly
- ☐ required intervention to prevent permanent impairment/damage
- ☐ other: ______

3 **Date of event** (mo/day/yr)

4 **Date of this report** (mo/day/yr)

5 **Describe event or problem**

6. **Relevant tests/laboratory data,** including dates

7 **Other relevant history, including preexisting medical conditions** (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1 **Name** (give labeled strength & mfr/labeler, if known)
#1
#2

2. **Dose, frequency & route used**
#1
#2

3. **Therapy dates** (if unknown, give duration)
from to (or best estimate)
#1
#2

4 **Diagnosis for use** (indication)
#1
#2

5. **Event abated after use stopped or dose reduced**
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6. **Lot #** (if known)
#1
#2

7. **Exp. date** (if known)
#1
#2

8 **Event reappeared after reintroduction**
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9 **NDC #** (for product problems only)
– –

10 **Concomitant medical products** and therapy dates (exclude treatment of event)

D. Suspect medical device

1 **Brand name**

2. **Type of device**

3. **Manufacturer name & address**

4 **Operator of device**
☐ health professional
☐ lay user/patient
☐ other:

5 **Expiration date** (mo/day/yr)

6.
model # ______
catalog # ______
serial # ______
lot # ______
other #

7. **If implanted, give date** (mo/day/yr)

8 **If explanted, give date** (mo/day/yr)

9 **Device available for evaluation?** (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ______ (mo/day/yr)

10 **Concomitant medical products** and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1 **Name & address**

phone #

2. **Health professional?**
☐ yes ☐ no

3. **Occupation**

4 **Also reported to**
☐ manufacturer
☐ user facility
☐ distributor

5. **If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box.** ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or **FAX to:**
1-800-FDA-0178

FDA Form 3500 (6/93)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

• 1-800-FDA-0178	to FAX report
• 1-800-FDA-7737	to report by modem
• 1-800-FDA-1088	to report by phone or for more information
• 1-800-822-7967	for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity, including the identity of a self-reporter, may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

The public reporting burden for this collection of information has been estimated to average 30 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send your comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to:

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Paperwork Reduction Project
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Washington, DC 20503

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U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service • Food and Drug Administration

FDA Form 3500-back

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The FDA Medical Products Reporting Program
Food and Drug Administration
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News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE Journal, New Products and Services Dept. 11030 Ables Lane, Dallas TX 75229-4593

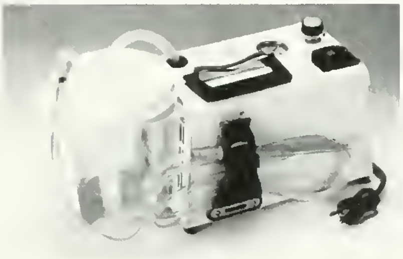
New Products & Services

FIT TESTING KIT. BioSafety Systems announces a Saccharin Fit Test Kit by Racal.® The company claims that the kit contains all the equipment necessary to conduct a qualitative fit test on tight-fitting respirators such as the N-95 and the HEPA. The kit includes a fit-test hood, fit-test collar, 3 bottles of taste threshold check solution, nebulizer for the fit-test solution, plastic hook and wire for cleaning the nebulizer, instructions and MSDS for Sodium Saccharin. Instructions and blank forms are printed in English and Spanish. A durable polyethylene carrying case makes transportation and storing the kit easy. For a free respiratory safety catalog, circle reader service card number 180.

NASAL SPRAY. GlaxoWellcome offers FLONASE® (fluticasone propionate) nasal spray, for the relief of nasal stuffiness, runny nose, and sneezing experienced by seasonal nasal allergy sufferers. According to GlaxoWellcome, the product, an aqueous, once-a-day, prescription nasal spray, works by reducing the inflammation of the nasal passages, thereby relieving nasal stuffiness. FLONASE is indicated for the treatment of both seasonal and perennial allergic rhinitis in patients aged 12 years and older. For a product information sheet and more information about indications and contraindications, circle reader service card number 181.

TREADMILL. Biodex Medical announces the RTM3 treadmill for cardiopulmonary rehabilitation. According to Biodex, the treadmill features 4 preset, automatic programs that allow the user to customize and store up to 8 user-defined programs. The RTM3 is designed for cardiac and pulmonary rehabilitation and allows patients to walk, jog, or run at any of 5 effort levels. The treadmill includes 3 handrails to accommodate patients of various heights, an Exact-Track™ belt system that maintains the belt in the center of the bed at all times, incline and decline settings, and a digital control panel that displays time, distance, work rate, speed, elevation, heart rate, and target heart rate. For details, circle reader service card number 182.

CATALOG. Laerdal Medical Corporation offers a free 1996 catalog. The 105-page book features emergency care products, systems, services, and training and technical support. To receive a catalog, circle reader service card number 185.



PORTABLE SUCTION UNIT. Armstrong Medical Industries Inc offers the S-Scort® DUET™ AC Suction Unit with battery back-up. According to Armstrong, the durable, self-contained unit provides effective suction with AC power or with the DUET emergency power supply. The DUET features a sealed lead battery with an internal dual-mode charger and is protected against overcharge and deep discharge. A variable regulator allows adjustable suction for adult and pediatric patients. The unit is recommended for emergency-cart, intrahospital-transport, and ambulance use. For more information, circle the reader service card number 183.

INSTANT COLD PACK. Florida Medical Industries Inc announces the TransCool™ Blood Sample Transporter Instant Cold Pack. The TransCool™ is a single use, disposable, and instant-cold product used to ensure short-term preservation and protection of blood samples while in transit to a laboratory. According to the company, the pouch holds up to 2 blood vials and remains cold (below 50° F) for more than 30 minutes. To request more information from Florida Medical, circle reader service card number 184.

RESPIRATORY CARE

Manuscript Preparation Guide

General Information

RESPIRATORY CARE welcomes original manuscripts related to respiratory care and prepared according to these Instructions. Manuscripts are blinded and reviewed by professionals who are experts in their fields. Authors are responsible for all aspects of the manuscript and receive galleys to proofread before publication. Each accepted manuscript is copyedited so that its message is clear and it conforms to the Journal's style. Published papers are copyrighted by Daedalus Inc and may not be published elsewhere without permission.

Editorial consultation is available at any stage of planning or writing. On request, specific guidance is provided for all publication categories. These Instructions and related materials are available. Write to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593, call (214) 243-2272, or fax (214) 484-6010.

Publication Categories & Structure

Research Article: A report of an original investigation (a study). It includes a Title Page, Abstract, Introduction, Methods, Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions.

Evaluation of Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification. It has a Title Page, Abstract, Introduction, Description of Device/Method/Technique, Evaluation Methods, Evaluation Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions.

Case Report: A report of a clinical case that is uncommon, or was managed in a new way, or is exceptionally instructive. All authors must be associated with the case. A case-managing physician must either be an author or furnish a letter approving the manuscript. Its components are Title Page, Abstract, Introduction, Case Summary, Discussion, References, Tables, Figures, and Figure Captions.

Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a pertinent topic that has been the subject of at least 40 published research articles. Title Page, Outline, Introduction, Review of the Literature, Summary, Acknowledgments, References, Tables, Appendices, and Figures and Captions may be included.

Overview: A critical review of a pertinent topic that has fewer than 40 published research articles.

Update: A report of subsequent developments in a topic that has been critically reviewed in this Journal or elsewhere.

Point-of-View Paper: A paper expressing personal but substantiated opinions on a pertinent topic. Title Page, Text, References, Tables, and Illustrations may be included.

Special Article: A pertinent paper not fitting one of the foregoing categories may be acceptable as a Special Article. Consult with the Editor before writing or submitting such a paper.

Editorial: A paper drawing attention to a pertinent concern; it may present an opposing opinion, clarify a position, or bring a problem into focus.

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PFT Corner: Like Blood Gas Corner, but involving pulmonary function tests.

Cardiorespiratory Interactions. A case report demonstrating the interaction between the cardiovascular and respiratory systems. It should be a patient-care scenario; however, the case—the central theme—is the systems interaction. CRI is characterized by figures, equations, and a glossary. See the March 1996 issue of RESPIRATORY CARE for more detail.

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Center main section headings on the page and type them in capital and small letters (eg, Introduction, Methods, Results, Discussion). Begin subheadings at the left margin and type them in capital and small letters (eg, Patients, Equipment, Statistical Analysis).

References. Cite only published works as references. Manuscripts accepted but not yet published may be cited as references: designate the accepting journal, followed by (in press). Please provide 3 copies of the in-press article for reviewer inspection. Cite references in the text with superscript numerals. Assign numbers in the order that references are first cited. On the reference page, list the cited works in numerical order. Follow the Journal's style for references. Abbreviate journal names as in *Index Medicus*. List all authors.

Article in a journal carrying pagination throughout volume:

1. Rau JL, Harwood RJ. Comparison of nebulizer delivery methods through a neonatal endotracheal tube: a bench study. Respir Care 1992;37(11):1233-1240.

Article in a publication that numbers each issue beginning with Page 1:

2. Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

3. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

4. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided. Those more than 3 years old should not be cited.)

5. Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11): 1087-1088.

Editorial in journal:

6. Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

7. Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

8. Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Paper accepted but not yet published:

9. Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (For any book, specific pages should be cited whenever possible.)

10. DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura, 1990:76-85.

Corporate author book:

11. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO. Publishing Sciences Group, 1977.

Chapter in book with editor(s):

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Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space-available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of the month two months preceding the month in which you wish the ad to run. Submit copy and insertion orders to Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

AARC & AFFILIATES

April 10–12—Louisiana Society
26th Annual Educational Meeting, "Respiratory Gumbo," at the Crown Sterling Suites, Baton Rouge, Louisiana. Exhibits, crawfish boil, and Pelican Bowl competition.
CRCE: Credit requested.
Contact: Jim Lanoha at (504) 381-6542.

April 11–12—Pineywood's Chapter of the Texas Society
8th Annual Seminar, Lufkin, Texas.
Contact: Sheila Sanchez at (409) 639-7006.

April 17–18—Utah Society
Annual convention at the Utah State Fair Park in the Grand Building, Salt Lake City, Utah. Dr Forrest Bird, keynote speaker.
Contact: Weber State University's Respiratory Care Department at (801) 626-7071.

April 17–19—Alabama Society
Annual Respiratory Care Conference, "1996 Educational Olympics," at the Radisson Hotel, Birmingham, Alabama. AARC President Charlie Brooks, keynote speaker. ASRC Sputum Bowl, Respiratory Olympics, and exhibits.
CRCE: Credit requested.
Contact: Karla Gray at (334) 705-3790.

April 17–19—Kansas Society
19th Annual Education Seminar, "Somewhere Over the Rainbow Is Clinical Care," at the Lawrence Holidome, Lawrence, Kansas.
CRCE: Credit requested.
Contact: Pat Munzer at (913) 231-1010, ext. 1284.

April 18–19—California Society
"Bright Horizons in Respiratory Care, Chapter 2" at the Hyatt Regency Suites, Palm Springs, California.
CRCE: Credit requested.
Contact: Tom Taylor at phone/fax number (909) 777-3214.

April 24–25—Connecticut Society
"Super Symposium" at the Four Points Sheraton, Waterbury, Connecticut.
Contact: Kevin Kenny at (203) 545-3212; Kathy Glenn-Nadal at (800) 882-0001, ext 42.

April 24–26—North Dakota Society
Annual education symposium, Bismarck, North Dakota. Keynote speaker: Patrick Reynolds.
CRCE: Credit requested.
Contact: Beth Hughes or Mike Runge at (701) 224-7870.

April 25–26—Arkansas Society
Diamond Conference, Arkansas Children's Hospital, Little Rock, Arkansas.
Contact: Mike Anders or Kathy Boyle, Respiratory Care Services, Arkansas Children's Hospital, Slot 303, 800 Marshall St, Little Rock AR 72202, (501) 320-3535, fax (501) 320-3411.

April 30–May 3—Michigan Society
Annual meeting, "Crossroads of Opportunity," at the Hyatt Regency, Dearborn, Michigan. Basic Life Support Instructor course.
Contact: MSRC office at (517) 336-7605.

April 30–May 3—Kentucky, Ohio, & Indiana Societies
1996 "Region II for Respiratory Care" Annual Meeting, "The Big Picture," at the Albert B Sabin Convention Center in the Hyatt Regency Cincinnati, Ohio.
Contact: Rhonda Windhorst, Chairperson, Health Care Partners, 10500 Bluegrass Pkwy, Louisville KY 40299, or call and leave a message at (800) 691-3046, mailbox 4.

May 1–3—Missouri Society
25th Annual Meeting, "Follow the Yellow Brick Road," at the TanTara Resort, Osage Beach, Missouri. Sputum Bowl preliminaries.
Contact: Donna Wemhoff at (314) 882-6491.

May 1–3—Montana Society
22nd Annual Convention at the Holiday Inn, Bozeman, Montana. Speaker: Jill Eicher, AARC director of state government affairs.
Contact: Dave Bunkers at (406) 657-4075.

May 1–3—South Dakota Society
Annual meeting at the Howard Johnson, Sioux Falls, South Dakota. Dr Forrest Bird, keynote speaker.
CRCE: Credit requested.
Contact: Mary Reinesch at (605) 333-6477.

May 7–9—Nebraska Society
Annual Great Plains Conference for Respiratory Care at the New Tower Inn, Omaha, Nebraska. AARC President-Elect Kerry George, keynote speaker.
Contact: Sue Waggoner at (402) 559-9503.

May 15–17—Southwest Region of the Texas Society
"25 Years of Excellence" Seminar at the El Paso Airport Hilton, El Paso, Texas.
CRCE: Credit requested.
Contact: Maria Soto, Mobile Diagnostics, at (915) 593-3519.

May 18–21—Washington Society
23rd Annual Pacific Northwest Regional Respiratory Care Conference at Cavanaugh's Inn at the Park, Spokane, Washington.
Contact: Bob Bonner, Chair of the Health, Education and PE Division, Highline Community College, PO Box 98000, Des Moines WA 98198, (206) 878-3710, ext 3469.

May 21—AARC Video Conference
"Managing Demand for Respiratory Care Services," Part 3 of the 1996, 7-part video conference, "Professor's Rounds in Respiratory Care" from 12:30 to 2 PM Eastern Time. Live telephone question-and-answer session airs June 10.
CRCE: 1 credit hour.
Contact: AARC at (214) 243-2272.

May 21–23—Arizona Society
Annual Education Seminar at the Phoenix Civic Plaza in Phoenix, Arizona.
Contact: ASRC, 3900 E Camelback Rd, Suite 200, Phoenix AZ 85018, (602) 912-5300.

May 24—Vermont/New Hampshire Society
"CE (continuing education) Cruise…IV" aboard the Motor Vessel Mount Wash-

ington, Weirs Beach, New Hampshire.
CRCE: Credit requested.
Contact: Bill Hay at (603) 267-7406.

May 24—Alamo District of the Texas Society
1st Annual Riverwalk Respiratory Symposium at the Marriott Riverwalk, San Antonio, Texas. Sponsored by: University of Texas Health Science Center at San Antonio, TSRC (Alamo District), & Wilford Hall Medical Center.
CRCE: Credit requested.
Contact: University of Texas Health Science Center at San Antonio, Department of Respiratory Care, 7703 Floyd Curl Dr, San Antonio TX 78284-7784, (210) 567-3706.

June 10–14—Florida Society
1996 Sunshine Seminar at the Fountain Blue Hilton Resort & Towers, Miami Beach, Florida.
Contact: Deborah Herrick at (305) 587-5010; fax (305) 583-2927.

June 19–21—Oklahoma Society
31st Annual Seminar, Oklahoma City, Oklahoma.
Contact: Gennie Ridlen at (405) 680-2154.

July 9—AARC Video Conference
"Capturing Respiratory Services Outside the Hospital Part I: Home Care," Part 4 of the 1996, 7-part "Professor's Rounds in Respiratory Care," from 12:30 to 2 PM Eastern Time. Live telephone question-and-answer session on July 29.
CRCE: 1 credit hour.
Contact: AARC at (214) 243-2272.

July 17–19—Texas Society
Annual convention, "Upgrade Your Memory," at the Wyndham Anatole Hotel, Dallas, Texas.
CRCE: Credit requested.
Contact: TSRC at (214) 680-2454.

OTHER MEETINGS

April 25–26—California Society for Pulmonary Rehabilitation
7th Annual Conference, "Cruising the Changing Currents: Facing New Challenges in Pulmonary Rehabilitation," at the Sutton Place Hotel, Newport Beach, California.
Contact: Jim Barnett at (714) 365-2106; Ray Alfonso at (714) 456-5218.

May 9–10—American Lung Association of Nevada
11th Annual Respiratory Health Conference at the Tropicana Resort & Casino, Las Vegas, Nevada.
Contact: American Lung Association of Nevada at (702) 431-6333.

May 10—Southwest Texas State University
2nd Annual Respiratory Care Symposium, "Managing New Frontiers in Respiratory Care," via interactive television from San Marcos to Laredo, Texas.
CRCE: Credit requested.
Contact: Lynda Harkins at (512) 245-8243.

May 10–15—American Thoracic Society/American Lung Association
1996 International Conference at the New Orleans Convention Center, New Orleans, Louisiana.
Contact: ATS/ALA International Conference, 1740 Broadway, New York NY 10019-4374, (212) 315-8700.

November 10–14—Asia Pacific Association for Respiratory Care
5th Congress of APARC at the Bangkok Convention Centre and Central Plaza Hotel, Bangkok, Thailand. Abstracts must be submitted by July 31.
Contact: Congress Secretariat, 5th Congress of APARC, Congress & Convention Experts (Thailand) Co Ltd, PO Box 10-163, Ladphrao, Bangkapi, Bangkok 10310, THAILAND.

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D. ☐ Staff Technician
E. ☐ Staff Therapist
F. ☐ Educator
G. ☐ Medical Director
H. ☐ Anesthesiologist
I. ☐ Pulmonologist
J. ☐ Other MD
K. ☐ Nurse

V. Are you a member of the AARC?
1. ☐ Yes 2. ☐ No

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For more information about how ASSESS can help you deliver better asthma care, **call HealthScan Products at 1-800-962-1266.**

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Setting the standard for peak flow monitoring.

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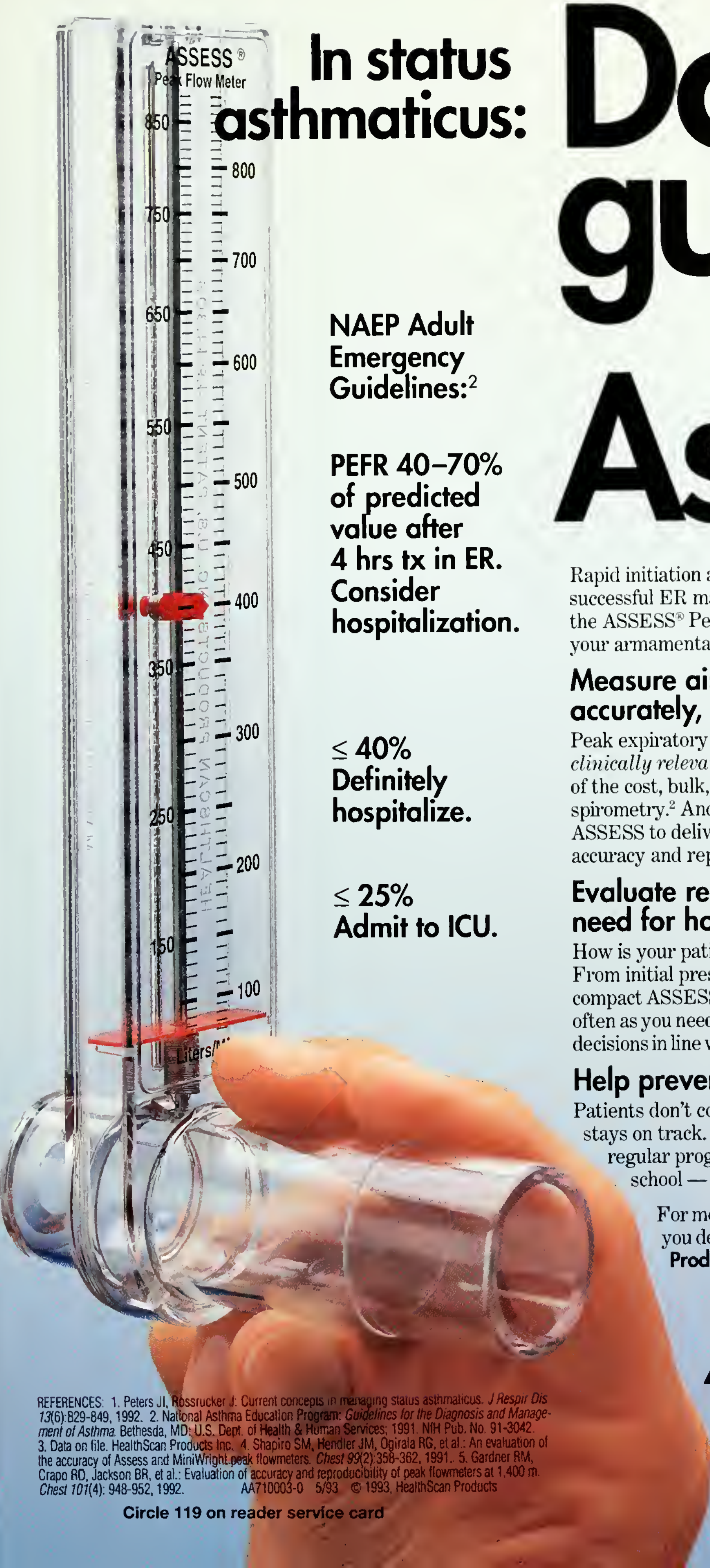

In status asthmaticus: Don't guess. Assess.

NAEP Adult Emergency Guidelines:[2]

PEFR 40–70% of predicted value after 4 hrs tx in ER. Consider hospitalization.

≤ 40% Definitely hospitalize.

≤ 25% Admit to ICU.

Rapid initiation and close monitoring of therapy are vital to successful ER management of severe asthma.[1] That's why the ASSESS® Peak Flow Meter should be a vital part of your armamentarium. ASSESS lets you —

Measure airway obstruction easily, accurately, cost-effectively.

Peak expiratory flow rate (PEFR) provides an objective, *clinically relevant* measurement of airflow — at a fraction of the cost, bulk, and inconvenience of conventional spirometry.[2] And when seconds count, you can count on ASSESS to deliver those measurements with superior accuracy and reproducibility.[3-5]

Evaluate response to therapy and need for hospitalization.

How is your patient doing? What should *you* be doing? From initial presentation through discharge, the rugged, compact ASSESS gives you the hard data you need — as often as you need it — to help you make informed treatment decisions in line with NAEP recommendations.[2]

Help prevent future acute exacerbations.

Patients don't come back when their maintenance therapy stays on track. Keep it on track with ASSESS — and a regular program of PEFR monitoring at home, work, or school — as a routine part of your discharge orders.

For more information about how ASSESS can help you deliver better asthma care, **call HealthScan Products at 1-800-962-1266.**

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